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ROCKY MOUNTAIN ARSENAL BASIN F LIQUID STORAGE TANK SPILL RISK ASSESSMENT

Prepared for
U.S. Army Corps of Engineers
Omaha District
Omaha, Nebraska
April 1, 1993

Woodward-Clyde

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THE SCOPE OF THIS RA IS LIMITED TO THE EVALUATION OF POTENTIAL HUMAN HEALTH RISKS ASSOCIATED WITH A FAILURE EVENT OF A TANK CONTAINING BASIN F LIQUID. THE REPORT CONTAINS NINE SECTIONS. SECTION 1.0 CONTAINS AN INTRODUCTION TO THE HEALTH RISK ASSESSMENT. SECTION 2.0 DESCRIBES THE SITE HISTORY, LOCATION AND LAND USE. SECTION 3.0 PROVIDES A BRIEF DESCRIPTION OF EXPOSURE PATHWAYS AND POTENTIAL RECEPTORS. SECTION 4.0 DESCRIBES THE SOURCES OF DATA USED AND IDENTIFIES CHEMICALS OF CONCERN. SECTION 5.0 DISCUSSES THE TOXICITY OF THE CHEMICALS OF CONCERN. SECTION 6.0 EXPLAINS THE METHODS USED FOR CALCULATION OF CARCINOGENIC RISKS AND THE NONCARCINOGENIC HAZARD INDEXES. SECTION 7.0 DESCRIBES UNCERTAINTIES INHERENT IN THE CURRENT METHODOLOGY USED TO DETERMINE POTENTIAL HUMAN HEALTH RISKS. SECTION 8.0 PRESENTS A SUMMARY OF RESULTS AND CONCLUSIONS. SECTION 9.0 INCLUDES THE REFERENCES CITED.

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TABLE OF CONTENTS

Section			Page
1.0	INTRODUCTION		1-1
	1.1	OBJECTIVES	1-1
	1.2	SCOPE	1-2
	1.3	REPORT ORGANIZATION	1-2
2.0	BACKGROUND		2-1
	2.1	SITE LOCATION	2-1
	2.2	HISTORY OF BASIN F LIQUID STORAGE	2-1
	2.3	CHEMICAL CHARACTERISTICS OF BASIN F LIQUID	2-2
	2.4	ON-SITE LAND USE	2-3
	2.5	OFF-SITE LAND USE	2-3
3.0	EXPOSURE PATHWAYS		3-1
	3.1	CHEMICAL SOURCES, RELEASE MECHANISMS, AND TRANSPORT MEDIA	3-1
	3.2	POTENTIAL HUMAN CONTACT AND RECEPTORS	3-3
	3.3	INTAKE ROUTES	3-3
4.0	ESTIMATING CHEMICAL INTAKES		4-1
	4.1	CHEMICALS OF CONCERN	4-1
	4.2	EXPOSURE POINT CONCENTRATIONS	4-2
	4.3	ESTIMATING CHEMICAL INTAKES	4-4
		4.3.1 Quantifying Average and Reasonable Maximum Exposures	4-5
		4.3.2 Calculation of Intake Factors	4-6
	4.4	EXPOSURE ASSUMPTIONS	4-6
5.0	TOXICITY ASSESSMENT		5-1
	5.1	TOXICITY ASSESSMENT FOR NONCARCINOGENIC EFFECTS	5-2
	5.2	TOXICITY ASSESSMENT FOR CARCINOGENIC EFFECTS	5-5

TABLE OF CONTENTS (Continued)

Section			Page
6.0	RISK CHARACTERIZATION		6-1
	6.1	METHODOLOGY FOR QUANTITATIVE RISK ESTIMATION	6-1
		6.1.1 Hazard Index For Noncarcinogenic Effects	6-1
		6.1.2 Carcinogenic Risk	6-3
		6.1.3 Risk Quantification	6-4
	6.2	RISK CHARACTERIZATION	6-5
7.0	UNCERTAINTIES AND LIMITATIONS		7-1
	7.1	DATA COLLECTION AND EVALUATION	7-1
	7.2	EXPOSURE ASSESSMENT	7-2
	7.3	TOXICITY ASSESSMENT	7-3
	7.4	RISK CHARACTERIZATION	7-5
8.0	SUMMARY OF RESULTS		8-1
9.0	REFERENCES		9-1

LIST OF TABLES

TABLE 4-1	CHEMICAL CHARACTERIZATION DATA OF BASIN F LIQUID
TABLE 4-2	RME CONCENTRATIONS
TABLE 4-3	AVERAGE CONCENTRATIONS
TABLE 4-4	LARGE STORAGE TANK FAILURE INTAKE ASSUMPTIONS FOR VOC INHALATION, HYPOTHETICAL RESIDENTS
TABLE 4-5	LARGE STORAGE TANK FAILURE INTAKE ASSUMPTIONS FOR INHALATION OF VOCs, ON-SITE WORKER
TABLE 4-6	DAY TANK FAILURE INTAKE ASSUMPTIONS FOR VOC INHALATION, HYPOTHETICAL RESIDENTS
TABLE 4-7	DAY TANK FAILURE INTAKE ASSUMPTIONS FOR INHALATION OF VOCs, ON-SITE WORKER
TABLE 5-1	EPA WEIGHT-OF-EVIDENCE CARCINOGENIC CLASSIFICATION OF CONTAMINANTS
TABLE 5-2	SUBCHRONIC RfDs AND SLOPE FACTORS FOR CHEMICALS OF CONCERN
TABLE 5-3	SHORT-TERM EXPOSURES OF POTENTIAL CONCERN
TABLE 5-4	RMA LARGE STORAGE TANK RME CONCENTRATIONS
TABLE 5-5	RMA DAY STORAGE TANK RME CONCENTRATIONS
TABLE 6-1	CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARDS, RMA LARGE STORAGE TANK FAILURE
TABLE 6-2	CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARDS, RMA "DAY" STORAGE TANK FAILURE

TABLE OF CONTENTS (Concluded)

LIST OF FIGURES

FIGURE 2-1	SITE LOCATION MAP
FIGURE 2-2	STORAGE AND DRY TANK LOCATION MAP
FIGURE 3-1	DRY AND STORAGE TANK AREA CONCEPTUAL SITE MODEL

LIST OF APPENDIXES

APPENDIX A	SPILL SCENARIO ASSUMPTIONS AND CALCULATIONS
APPENDIX B	INTERPRETATION OF ASSESS OUTPUT AND ASSESS REPORTS
APPENDIX C	TOXICITY PROFILES

1.0 INTRODUCTION

The Program Manager for the Rocky Mountain Arsenal (PMRMA) is implementing the final disposal of the liquid removed from the former Basin F surface impoundment. The Basin F liquid has been characterized and a remedial alternative has been selected for its final treatment and disposal. A Submerged Quench Incinerator (SQI) has been constructed for incineration of the liquid. The Basin F liquid is currently stored in three large storage tanks and one covered pond at the Rocky Mountain Arsenal (RMA).

The following human health risk assessment (RA) has been prepared to address potential human health risks associated with hypothetical accidental release of the entire contents of Basin F liquid from one of the large storage tanks or a smaller "day" storage tank associated with the SQI facility.

1.1 OBJECTIVES

The primary objective of this RA was to use recently collected data to evaluate potential human health risks associated with a catastrophic failure event of a tank containing Basin F liquid. Where possible, this RA used site-specific data. This was consistent with the recently issued guidance by the United States Environmental Protection Agency (EPA) Risk Assessment Council stressing the use of site-specific data to evaluate potential health risks (EPA 1992a,b). Another objective was to follow the Risk Assessment Council's guidance for risk characterization limiting discussion of information to risk assessment instead of including any risk management discussions, identifying key uncertainties and their potential to under- or overestimate risk, and including information on a range of exposure. While the reasonable maximum exposure (RME) is designed to be a measure of "high-end" exposure and is used by risk managers in making remedial decisions (if any) to achieve protection against risk to human health, the average exposure may be used for informational purposes to aid in discussing uncertainties (EPA 1992c). Conservative assumptions of exposures were used when site-specific data were lacking or incomplete.

1.2 SCOPE

The scope of this RA is limited to the evaluation of potential human health risks associated with a failure event of a tank containing Basin F liquid. The approach used is based on EPA guidance for the evaluation of human health risks at Superfund sites. Specifically, this risk assessment is consistent with guidance provided in the following EPA documents: Exposure Factors Handbook (EPA 1989a), Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (EPA 1989b), and the Integrated Risk Information System (IRIS) (EPA 1992d), a computer database containing up-to-date health risk and EPA regulatory information for numerous chemicals. EPA cautions that these resources are intended only to provide guidance in the conduct of health risk assessments and that considerable professional judgment must be used in their application (EPA 1989b).

1.3 REPORT ORGANIZATION

Section 1.0 of this report contains an introduction to the health risk assessment including the objectives and scope. Section 2.0 describes the site history and location. Land use in the area is also discussed. Section 3.0 provides a brief description of exposure pathways and potential receptors. Section 4.0 presents a brief description of the sources of data used and identifies chemicals of concern. Air modeling used to estimate exposure point concentrations is also described. Estimates of intake for each chemical of concern are also presented in this section.

Section 5.0, the Toxicity Assessment, describes the toxicity of the chemicals of concern and how toxicity values are applied in the quantification of potential health risks and hazards. Section 6.0, Risk Characterization, explains the methods used for calculation of carcinogenic risks and the noncarcinogenic hazard indexes. This section also presents a summary of the calculated potential health risks for each complete exposure pathway and receptor population. Section 7.0 describes uncertainties inherent in the current methodology used in the determination of potential human health risks. In addition, site-specific uncertainties and limitations are identified and discussed. A summary of results and conclusions is presented in Section 8.0. Section 9.0 includes the references cited.

2.0
BACKGROUND

2.1 SITE LOCATION

RMA is located in Adams County, Colorado, approximately 10 miles northeast of downtown Denver. The site occupies 16,914 acres (Weston 1991). The site location is shown in Figure 2-1. The storage and day tank areas are shown in Figure 2-2.

2.2 HISTORY OF BASIN F LIQUID STORAGE

RMA was established in 1942 and has been the site of chemical and incendiary munitions manufacturing, and chemical munitions demilitarization. Industrial chemicals were manufactured at RMA from 1947 to 1982. Disposal practices at RMA have included routine discharge of industrial and munitions waste effluents to evaporation basins.

In 1956, Basin F was constructed in the northern part of RMA in Section 26. Basin F had a surface area of 92.7 acres and a capacity of approximately 243 million gallons. The basin was created by construction of a dike around a natural depression and was lined with a 3/8-inch catalytically blown asphalt membrane. A protective earthen blanket approximately 1 foot thick was placed on top of the membrane. A vitrified clay pipe with chemically resistant sealed joints was installed between Basin F and the facilities where the wastes were generated. From August 1957 until its use was discontinued in December 1981, Basin F was the only evaporative disposal facility in service at RMA.

In 1986, the Department of the Army, Shell Oil Company, and the EPA Region VIII agreed that an accelerated remediation be conducted to contain the liquid and contaminated soils in and under Basin F. This remediation was undertaken pursuant to the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA).

The first Interim Response Action (IRA-1) of the remediation involved the transfer of Basin F liquid to three lined storage tanks (Tanks 101, 102, and 103) and one covered pond of a surface impoundment facility. Transfer of Basin F liquid to tanks for interim storage was initiated in May 1988. Approximately 4 million gallons of liquid is presently stored in the tanks, 1.3 million gallons in each tank. The pond contains approximately 7 million gallons.

The second Interim Response Action (IRA-2) for Basin F liquid addresses treatment and disposal of the contents of the storage tanks and the pond. This IRA was initiated in September 1988 and is in progress. Further characterization of the stored liquid, selection of a treatment alternative for the liquid, pilot scale demonstration of the selected treatment technology, and detailed engineering design and construction of the remedial treatment process have been performed. Thermal treatment, in the form of the SQI, has been selected as the remedial treatment process. The SQI facilities include two 14,000 gallon "day" tanks (Tanks 105 and 106) for temporary storage of the Basin F liquid before it is fed to the SQI system.

2.3 CHEMICAL CHARACTERISTICS OF BASIN F LIQUID

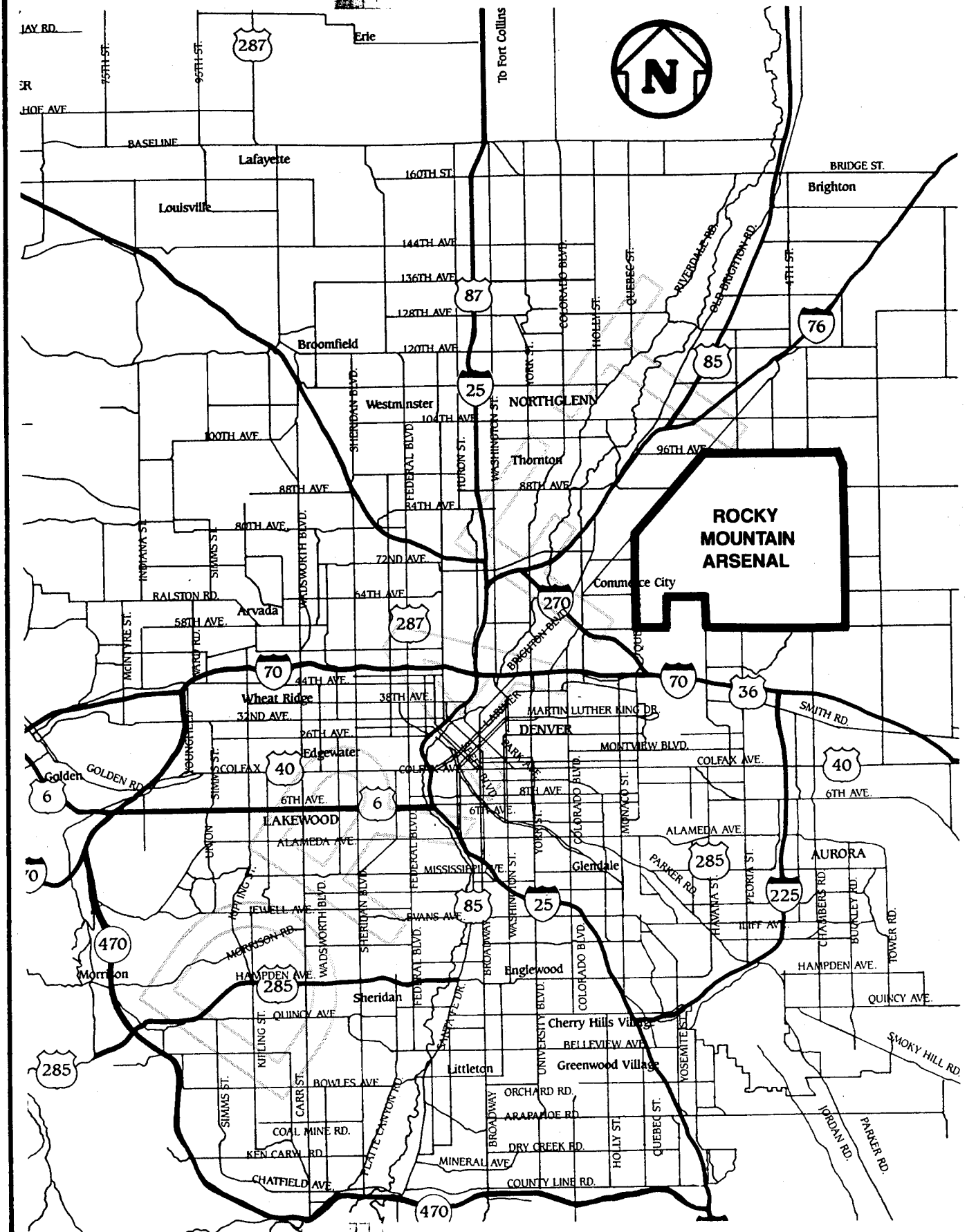
Basin F liquid is an aqueous liquid containing a complicated mixture of hydrocarbons, chlorinated hydrocarbons, salts, metals, and other process intermediates, by-products, and wastes. It is known that quantities of ammonium phosphate, and later copper sulfate, were added to Basin F at different times. Numerous studies conducted to characterize Basin F liquid indicate that its contents include alcohols, fluoride, chloride, insecticides, chlorinated organics, chlorophenylmethylsulfone, pesticides, chlorophenylmethyl sulfoxide, phenols, dicyclopentadiene, phosphorous, p,p-DDE, p,p-DDT, sulfate, acetophenone, aldrin, isodrin, arsenic, mercury, chlorophenylmethyl sulfone, metals, pentachloroethane, dibromochloropropane, tetrachloroethene, dithiane, toluene, dieldrin, trichloroethane, xylene, dimethyl methylphosphonate (DMMP), endrin, and diisopropylmethyl phosphonate (DIMP).

2.4 ON-SITE LAND USE

The current land use at RMA is a combination of industrial, commercial, recreational, and natural habitat. The industrial and commercial classifications relate to the nature of the on-site buildings: an army administration building, a fire department, a post office, groundwater treatment facilities, and a rail classification yard (Weston 1991). The recreational classification stems from the occasional "catch and release" fishing (Ebasco 1990) from several lakes onsite (Weston 1991). The majority of the site, including the bald eagle management area, is classified as a natural habitat for wildlife (Weston 1991).

2.5 OFF-SITE LAND USE

Land usage surrounding RMA is characterized by a varied pattern of heavy and light industrial, residential, and agricultural. The north and east are predominantly agricultural, the south is primarily heavy industry, and the west is a combination of commercial and industrial. Residential areas are intermixed primarily to the west and south, but also to the northwest (Weston 1991). Stapleton International Airport, the fifth largest in the United States, occupies the most acreage near RMA. Construction of a new airport is under way to the east of RMA.



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Date :	3//30/1993

SITE LOCATION MAP

FIG. 2-1

3.0
EXPOSURE PATHWAYS

This section discusses the potential release and transport of chemicals from a tank failure, the exposure pathways by which the receptor populations may be potentially exposed to chemicals, and the specific receptors included in the quantitative evaluation.

An exposure pathway describes a specific environmental pathway by which an individual can be exposed to chemical constituents present at or originating from a source. A complete exposure pathway includes five necessary elements:

- A source of chemicals
- A mechanism of chemical release to the environment
- An environmental transport medium
- A point of potential contact
- An intake route

Each of these five elements must be present for an exposure pathway to be complete and for chemical intake to occur. Incomplete exposure pathways do not result in human exposures. Without exposure, there can be no risk; therefore, incomplete exposure pathways have not been included in the risk characterization process.

3.1 CHEMICAL SOURCES, RELEASE MECHANISMS, AND TRANSPORT MEDIA

Chemical constituents present in the Basin F liquid stored in the tanks have been identified as potential sources for chemical release. In the event of a catastrophic failure of one of the tanks, the stored Basin F liquid would spill into the secondary containment surrounding the tanks. Once exposed to the atmosphere, the volatile and semi-volatile compounds contained in the liquid could volatilize. The spill would be restrained from leaking to other media by secondary containment mechanisms. For example, the day tanks are on an epoxy-coated concrete pad that is bermed and drains to a collection sump. The large storage tanks are in an earthen-bermed area with a geomembrane interposed between the subsurface soil and a layer of soil atop the membrane itself.

This precludes any spillage from leaching through to the underlying soil and to groundwater and prevents surface run-off from carrying any spillage away from the area. Other than air, no other environmental media would be involved in the transport of chemical constituents from these sources.

Factors influencing the volatilization of the compounds from the spill include, but are not limited to, the surface area of the spill; the physical properties of the compounds, including vapor pressure; and meteorological conditions, particularly wind speed and precipitation. A secondary release mechanism, intrusive action, was considered as part of the cleanup effort. The action of cleaning up the spill may, to some small degree, enhance the rate of volatization; however, the degree would be relatively infinitesimal compared to the rate of total volatization.

To determine the length of time required for cleanup, hence, the time the spill would be exposed to the atmosphere, several assumptions were made. In the event of a 1.3-million gallon spill from the failure of a large storage tank, it was assumed that two vacuum trucks would be employed to remove the liquid from the secondary containment and transport it to Pond A. Using the capacity of the trucks with estimated loading, transport, and unloading times, it was estimated that it would take nine days to remove the spill.

In the event of a 14,000-gallon spill from a day tank, it was assumed that the 25-gallon per minute (gpm) pump in the secondary containment would be the only device used to remove the spill. Operating the pump full time would remove the spill in just under 10 hours. These calculations, along with calculations estimating the surface area of the spills, are outlined in Appendix A.

3.2 POTENTIAL HUMAN CONTACT AND RECEPTORS

RMA is a public-access restricted facility; therefore, points of potential human contact with the spill or its contaminants are limited. Workers involved in the spill clean-up were not considered receptors because these workers would be trained hazardous waste workers and would be protected by personal protective equipment and clothing as

prescribed by the Occupational Safety and Health Administration (OSHA). The restricted nature of the facility would prevent others from accessing the area.

With transportation of volatilized chemicals by wind dispersion, receptors at a distance must be considered. An office worker in the on-site administration building was considered a receptor for this scenario along with an adult and child resident at the fenceline of the RMA property. The on-site office worker was assumed to be located in the RMA administration building, Building 111, which is approximately 9,700 feet from the tanks containing Basin F liquid. The off-site residents were conservatively assumed to be located at the nearest RMA boundary, approximately 5,650 feet from the tanks.

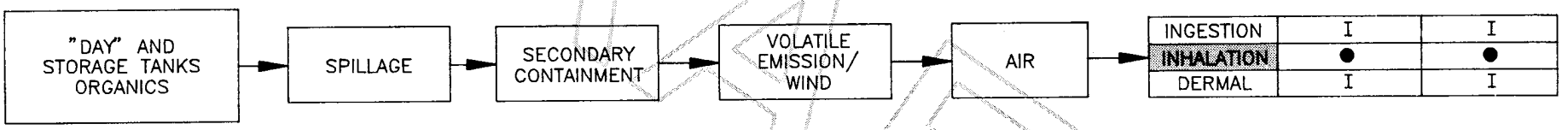
3.3 INTAKE ROUTES

The final component of a complete exposure pathway is an intake or exposure route into the body. A human exposure route is the manner by which a chemical is taken into the body. The three basic human exposure routes are dermal absorption, inhalation, and ingestion. With the release mechanisms and transport media limited to volatilization and wind dispersion, potential human contacts considered in this tank failure scenario include inhalation of the volatilized compounds and dermal absorption of chemicals contained in the vapors.

Dermal absorption of vapor phase chemicals is not considered significant by the EPA: the rate of absorption through the skin is thought to be lower than inhalation intakes, so this is not considered as a significant uptake route (EPA 1989b). Therefore, only inhalation of compounds volatilized from the spill was considered a complete pathway for the receptors.

A Conceptual Site Model (CSM) shows all potentially complete exposure pathways for a source (Figure 3-1). The significant exposure pathways are represented with a solid circle; an open circle represents those pathways in which contaminant intakes are considered to be relatively insignificant in comparison to other exposure pathways. EPA guidance defines an insignificant pathway as one that has an exposure estimated to be two or more orders of magnitude less than by other pathways (of the same receptor); a

pathway is also considered insignificant if the risks are much less for that pathway, or if the likelihood of exposure by that pathway is very small (EPA 1989b).



L E G E N D

- I INCOMPLETE EXPOSURE PATHWAYS
- ● SIGNIFICANT EXPOSURE PATHWAY
- O RELATIVELY INSIGNIFICANT EXPOSURE PATHWAY
- NA NOT APPLICABLE

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Date :	3/3/93

DAY AND STORAGE TANK AREA CONCEPTUAL SITE MODEL

ROCKY MOUNTAIN ARSENAL

FIG. 3-1

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4.0
ESTIMATING CHEMICAL INTAKES

4.1 CHEMICALS OF CONCERN

This step of the RA involved the review of existing Basin F liquid chemical data. The objective of the data analysis was to identify chemicals of concern (COCs) that were related to the exposure scenarios considered. The most comprehensive summary of historical and recent chemical characterization data for Basin F liquid was found in the Final Draft Human Health Risk Assessment for the RMA Basin F Incineration project report by Roy F. Weston (Weston 1991). Analytical data on the Basin F liquid stored in the tanks was collected by Scott (February 1989) and Weston (August 1989, February 1990, April 1990 and October 1990). In the Incineration RA (Weston 1991), the data collected from the tanks was compiled and the arithmetic mean and maximum values of the detected compounds were tabulated.

Volatile and semi-volatile organics were the compounds of interest for the relevant exposure scenarios. To be conservative, the compilation of all data collected from the tanks was used. These data provided an initial list of 13 volatile and 4 semi-volatile compounds that were detected, as shown in Table 4-1. However, it is clear that some of the organic compounds have degraded since the earlier analysis: the most recent data (October 1990) collected from the Basin F liquid stored in the tanks shows detection of only five volatile and semi-volatile compounds: 1,2 dichloropropane, chlorobenzene, methylene chloride, 4-chlorophenylmethylsulfone, and dimethyl methylphosphonate.

The toxicity values (slope factors and reference doses) were researched for the preliminary list of 17 compounds. The Integrated Risk Information System (IRIS), the EPA on-line database (EPA 1992d), was researched first. For some of the compounds, inhalation toxicity values were not available in IRIS, and the Health Effects Assessment Summary Table (HEAST) (EPA 1992e) was researched. The status of the toxicity values for the preliminary COCs is shown in Table 4-1. For many of the chemicals, no current inhalation toxicity values were available. With the exception of tetrachloroethene and trichloroethene, these chemicals were removed from the COC.

These two chemicals were retained on the COC list and the most recent (but currently withdrawn) toxicity values were used. The resulting COCs are as follows:

- 1,1-Dichloroethene
- 1,2-Dichloropropane
- Aldrin
- Chlorobenzene
- Chloroform
- Ethylbenzene
- Methylene Chloride
- Tetrachloroethene
- Toluene
- Trichloroethene

4.2 EXPOSURE POINT CONCENTRATIONS

An exposure point is a specific location where receptors can come in contact with site-related chemicals. Exposure points are usually selected so that the greatest and most reasonable potential exposures will be quantitatively evaluated. Evaluation of receptor risks at these points is conservative and therefore provides an upper bound on the potential risks for receptors at other exposure points not selected for quantitative evaluation. Exposure points included the nearest RMA boundary for the assessment of the residential receptors and the on-site administration building for the assessment of the on-site office worker.

Air quality modeling of exposure point concentrations from a release of Basin F liquids from either a day storage tank or a large storage tank was performed using the ALOHA model (NOAA 1988). ALOHA (Areal Location of Hazardous Atmospheres) was developed by the Hazardous Materials Response Branch of the National Oceanic and Atmospheric Administration for emergency response scenarios as part of a comprehensive package named CAMEO. The model contains a chemical library containing data of chemical properties that are pertinent to estimating resultant air emission rates. The model uses the chemical properties in conjunction with site-specific atmospheric conditions and source strength data to estimate the movement and

dispersion of the chemical release. The dispersion characteristics of the atmosphere are assumed to follow a Gaussian (normal) distribution. The nominal accuracy of the model is a factor of 2. For example, if the predicted concentration is 50 ppm at some distance from the source, then a measurement at that point has a high probability of being between 25 and 100 ppm. The wind patterns are assumed to be uniform throughout the length of the plume. It was assumed that the wind blew directly toward the receptors. The model does not take into account changes in wind speed and direction or small scale variations in the wind pattern caused by terrain effects or obstacles such as buildings.

The ALOHA model is run by identifying the COCs from the chemical library, selecting appropriate atmospheric conditions, and identifying source information. The chemical data in the library include the chemical name, molecular weight, vapor pressure, and boiling point. Atmospheric data used in the model include atmospheric stability, inversion height, wind speed, wind direction, air temperature, and ground roughness. The source information includes the volume of the liquid or the surface area of the puddle or ponded liquid.

When the model was used in the instantaneous release mode, it was assumed that the entire contents of the storage tank were released instantaneously. In this mode, the model was run using the measured maximum concentration of each chemical of concern in the Basin F liquid. The predicted results were then proportionally scaled using the average concentrations of each chemical in the Basin F liquid for the average exposure scenario. The model displays the predicted ambient air concentrations in the form of a "puff" which travels downwind from the source and disperses quickly. The ambient concentration of the chemical depends on the downwind distance of the receptor and the dispersion characteristics of the atmosphere.

In the puddle evaporation mode, the model predicts concentrations that would result from the evaporation of the puddle. This option assumes that the temperature of the puddle is equal to the ambient air temperature and that the released liquid is comprised of 100 percent of the chemical of concern. The predicted concentration of each chemical of concern was then proportionally scaled using the measured maximum and average concentrations of each chemical in the Basin F liquid.

After completing both the instantaneous release and the puddle evaporation modeling scenarios, the resultant predicted exposure point air concentrations for each scenario were combined using a time-weighted average of the modeled concentrations with the estimated residence time for these concentrations. It was estimated that the instantaneous release concentrations would occur over the receptor exposure points for approximately one minute and then the concentrations from the puddle evaporation would occur until the puddle had been removed (9 days total for the large storage tank and 10 hours for the day tank). These assumptions along with using stable atmospheric conditions and hot air temperatures in the model result in conservative or worst-case predicted concentrations.

The list of ten COCs along with the measured average and maximum concentrations of each chemical in the Basin F liquid was used in the model. Refer to Table 4-1 for a list of the maximum values and arithmetic means of the COCs. Atmospheric conditions chosen for each model run were stable atmospheric conditions (Stability Class F), low wind speed, a steady wind direction, summertime air temperature (the average July high temperature for Denver, Colorado was used), and the rural ground roughness option. Source data input into the model included volumes of the liquid in the large storage tank (1,333,000 gal) and the small tank (14,000 gal) used in the instantaneous release mode of the model, and the surface area of the puddle formed by the liquid released from the large tank (76,644 ft^2) and from the small tank (1,002 ft^2) used in the puddle evaporation mode of the model. The distances to the receptors were also input. Ambient air concentrations of each chemical of concern were estimated from the model.

Table 4-2 presents the modeled instantaneous, puddle, and time-weighted average ambient air concentrations for each chemical for the reasonable maximum exposure (RME) and the average exposure for the large storage tank failure event. Table 4-3 presents these concentrations for the day storage tank failure event.

4.3 ESTIMATING CHEMICAL INTAKES

The next step was to quantify the magnitude, frequency, and duration of exposures. Chemical intakes were calculated using guidance in Risk Assessment Guidance for Superfund (EPA 1989b) and Exposure Factors Handbook (EPA 1989a), other EPA

documents and scientific journal articles, site-specific information, and professional judgment regarding likely exposure conditions, as appropriate. Chemical intakes were estimated using the modeled average and reasonable maximum exposure (RME) chemical concentrations, reasonable estimates of exposure parameters such as body weight, inhalation rates, and other assumptions regarding frequency and duration of exposure. The intake factors used to quantify chemical intakes are presented in this section.

4.3.1 Quantifying Average and Reasonable Maximum Exposures

Intake of a chemical is generally described in units of milligrams (mg) of chemical per kilogram (kg) of body weight per day (mg/kg/day). The magnitude of exposure to a chemical (or intake) is a function of exposure point concentration and values that describe the exposed population (i.e., contact rate, exposure frequency and duration, body weight, etc.).

Each exposure variable can be described by a range of values. For purposes of this assessment, two measures of exposure were defined using two types of exposure concepts: (1) an average exposure, and (2) an RME. Average and RME values were estimated in accordance with guidance provided in EPA's Risk Assessment Guidance for Superfund (EPA 1989b). Average exposure was estimated using average values and best conservative estimates of exposure variables to characterize the population and their normal activity patterns. The RME was obtained by selecting maximum chemical concentrations at the exposure point and using the receptor population characteristics and activity patterns that would result in a maximum exposure reasonably expected to occur at the site. The intent of the RME is to conservatively estimate a chemical intake that is well above average but still within the range of possible exposures. For example, EPA guidance recommends that the 95 percent upper confidence limit (UCL) on the arithmetic mean concentration or the maximum concentration (whichever is lower) be used for characterizing the chemical concentration for the RME scenario (EPA 1989b). Since an UCL could not be calculated due to a relative paucity of recent data for COCs, the maximum concentrations were used for input into the air model. Generally, maximum concentrations are greater than 95 percent UCLs making for a more conservative estimate of exposure (EPA 1989b).

4.3.2 Calculation of Intake Factors

The intake factor is a value that combines the site-specific and receptor-specific assumptions for a given exposure pathway. The intake factor multiplied by the concentration of a chemical of concern results in an estimate of the chemical intake in mg/kg/day for that receptor population and exposure pathway. The generic equation for calculating intake is the following:

$$\text{Intake (mg/kg/day)} = \text{Concentration} \times \text{Intake Factor}$$

$$\text{Intake Factor} = \frac{IR \times EF \times ED \times AF}{BW \times AT}$$

where:

- IR = Inhalation Rate (mg/m^3)
- EF = Exposure Frequency (days/year)
- ED = Exposure Duration (years)
- AF = Absorption Factor (unitless)
- BW = Body Weight (kg)
- AT = Averaging Time (days)

The intake factor is multiplied by the concentration for a chemical of concern yielding the daily chemical intake. Separate intake factors were calculated for each scenario (average and RME) and receptor. A more detailed description of the values used for intake calculations is presented in Section 4.4.

4.4 EXPOSURE ASSUMPTIONS

The extent of potential intake received through the various exposure pathways is based on EPA guidance, site-specific information, and professional judgment. It is not possible to estimate the exact level of exposure for specific individuals due to uncertainties in behavior patterns and incomplete knowledge of exposure variable values. This risk assessment used site-specific data when available and conservative assumptions where necessary to not underestimate potential human exposures.

This risk assessment evaluates exposures to carcinogenic and noncarcinogenic chemicals of concern. The evaluation for noncarcinogenic effects uses a subchronic exposure because of the relatively short exposure times described below (EPA 1989b). The EPA recommends that when evaluating chemicals for noncarcinogenic effects, the intake should be calculated by averaging the intake over the duration of exposure (referred to here as averaging time or AT) (EPA 1989b). For carcinogenic effects, intakes were calculated by averaging the total chemical exposures during the exposure period over an average lifetime of 70 years. The use of different averaging times for estimating carcinogenic and noncarcinogenic intakes is based on currently held scientific opinion that the mechanism of action of carcinogens and noncarcinogens is different.

Children were identified as a sensitive receptor population. Therefore, exposure of both adult and child residential receptors was considered.

For inhalation exposure time associated with the day tank failure, it was assumed that the residential receptor would be exposed to COCs in the vapor phase for 10 hours for both average and RME scenarios. This is based on the estimated amount of time necessary to clean up total spillage from the smaller tank. It was assumed that the on-site worker would be exposed to COCs in the vapor phase for 8 hours for both average and RME scenarios in the event of a day tank failure. For the inhalation exposure time due to the failure of the large storage tank, 8 hours a day for 7 days was used for the on-site worker. Although the total cleanup time for the large tank failure has been estimated at 9 days, 7 days was used as the exposure duration for the on-site worker to account for one weekend within the 9-day cleanup time frame. Sixteen hours per day for nine days was used for the average scenario, and 24 hours a day for 9 days was used for the RME scenario adult and child off-site residents. This was based on the number of hours the receptors spent either at home or at work and the number of days to clean up the total spillage from the large tank. Twenty-four hours was used for the RME scenario to account for members of the community who may spend the entire day at home.

The assumption was made that the duration of receptor exposure to COCs would equal the cleanup times for each failure event. This assumption overestimates risk by using as steady-state concentrations for each COC. Despite all three receptors being indoors,

where spill-related COC concentrations would be lower than outdoors, modeled outdoor concentrations were used as exposure point concentrations. This assumption also overestimates risk.

Averaging times for noncarcinogenic effects were the total number of days over which exposure could occur for each scenario. Averaging times for carcinogenic effects were calculated by averaging the exposure over a lifetime or 25,550 days (EPA 1989b).

Exposure of human receptors to chemicals of potential concern through inhalation was estimated by consideration of the following variables: (1) the volume of air inspired (inhalation rate); (2) the exposure time, frequency and duration; and (3) pulmonary absorption from the inhaled air. The equation and specific values used to estimate intake of chemicals via inhalation are summarized in Tables 4-4 through 4-7.

Inhalation rates for the adult and child residents were 0.83 m^3/hr and 1.25 m^3/hr for average and RME scenarios, respectively, calculated from daily inhalation rates of 20 m^3/day and 30 m^3/day (EPA 1989a). The inhalation rate used for the on-site office worker was 0.83 m^3/hr for the average exposure and RME scenario (EPA 1989a).

TABLE 4-1
CHEMICAL CHARACTERIZATION DATA OF BASIN F LIQUID

Compounds	Arithmetic Mean* (mg/l) Tanks	Maximum Value* (mg/l) Tanks	Toxicity Value: Slope Factor (mg/kg-day)$^{-1}$ Inhalation	Slope Factor (mg/kg-day)$^{-1}$ Oral[a]	RfD (mg/kg-day) Inhalation	RfD (mg/kg-day) Oral[a]
Volatiles						
1,1,1-Trichloroethane	NA	NA				
1,1,2-Trichloroethane	NA	NA				
1,1-Dichloroethane	NA	NA				
1,1-Dichloroethene	2.24E-01	3.22E-01	●	○	P	P
1,2-Dichloroethane	NA	NA				
1,1-Dichloroethene (total)	2.05E-02	5.14E-02	ND	ND	ND	P
1,2-Dichloropropane	5.50E-01	1.10E+00	ND	○	●	ND
1,3-Dimethylbenzene[b]	7.16E-02	1.23E-01				
2-Chloroethylbenzene	NA	NA				
Acetone	1.93E+00	1.93E+00	ND	ND	ND	○
Ammonia	NA	NA				
Benzene	NA	NA				
Bromoform	NA	NA				
Bromomethane	NA	NA				
Carbon Tetrachloride	NA	NA				
Chlorobenzene[c]	3.95E-02	1.12E-01	R	R	●	○
Chloroform	2.57E-01	3.49E-01	●	○	P	○
Dicyclopentadiene	NA	NA				
Ethylbenzene	8.15E-02	1.63E-01	ND	ND	●	○
Methanol	4.73E+03	4.73E+03	ND	ND	P	○
Methylene Chloride	2.50E+00	1.10E+01	●	○	●	○
Tetrachloroethene	1.35E-01	1.50E-01	●	○	ND	○

TABLE 4-1
(Continued)

Compounds	Arithmetic Mean* (mg/l) Tanks	Maximum Value* (mg/l) Tanks	Toxicity Value: Slope Factor (mg/kg-day)$^{-1}$ Inhalation	Slope Factor (mg/kg-day)$^{-1}$ Oral[a]	RfD (mg/kg-day) Inhalation	RfD (mg/kg-day) Oral[a]
Toluene	2.34E-02	4.22E-02	ND	ND	●	○
Trichloroethene[d]	6.88E-02	1.10E-01	●	WD	WD	WD
Xylene (total)	2.61E-01	9.88E-01	ND	ND	R	○
Semivolatiles						
1,2,4-Trichlorobenzene	NA	NA				
1,4-Dichlorobenzene	NA	NA				
4-Chlorophenylmethylsulfone	1.35E+02	3.01E+02	ND	ND	ND	ND
4-Chlorophenylmethylsulfoxide	NA	NA				
4-Nitrophenol	NA	NA				
Acenaphthene	NA	NA				
Aldrin[e]	3.83E-01	1.00E+00	●	○	ND	○
Atrazine	NA	NA				
Cyanide	NA	NA				
Dieldrin	NA	NA				
Diisopropyl Methylphosphonate	NA	NA				
Dimethyl Methylphosphonate	2.80E+02	8.24E+02	ND	ND	ND	P
Dimethyldisulfide	8.40E+01	8.40E+01	ND	ND	ND	ND
Dithiane	NA	NA				
Endrin	NA	NA				
Hexachlorocyclopentadiene	NA	NA				
Isodrin	NA	NA				
Malathion	NA	NA				

TABLE 4-1
(Concluded)

Compounds	Arithmetic Mean* (mg/l) Tanks	Maximum Value* (mg/l) Tanks	Toxicity Value: Slope Factor (mg/kg-day)$^{-1}$ Inhalation	Slope Factor (mg/kg-day)$^{-1}$ Oral[a]	RfD (mg/kg-day) Inhalation	RfD (mg/kg-day) Oral[a]
Parathion	NA	NA				
Pyrene	NA	NA				
Supona	NA	NA				
Urea	NA	NA				
Vapona	NA	NA				
ppDDE	NA	NA				
ppDDT	NA	NA				

*Source : Weston 1991.

ND = Not determined; no data on IRIS
WD = Withdrawn from IRIS as of February 26, 1993
NA = Not analyzed
● = Toxicity value used in quantitative risk assessment; taken from IRIS (EPA 1993)
○ = Toxicity value present but oral route not considered
P = Pending on IRIS as of February 26, 1993
R = Under review

[a] Oral route shown here only for convenience; the oral pathway is an incomplete exposure pathway and is not quantitatively evaluated (see Section 4.0).
[b] Same as m-xylene (see xylenes total)
[c] The inhalation RfD taken from Table 2 of HEAST FY92.
[d] Slope factor withdrawn from IRIS as of 2/93, slope factor from HEAST FY91 used.
[e] Route-to-route extrapolation; SF from inhalation unit risk of 4.9E $\mu g/m^3$. This unit risk should not be used if air concentration exceeds 2 $\mu g/m^3$ as it may be inappropriate (EPA 1993, IRIS)

▒▒ = chemicals of concern quantitatively evaluated in the risk assessment

TABLE 4-2

RMA LARGE STORAGE TANK
MODELED AIR CONCENTRATIONS

RME Concentrations mg/m3

Chemical	Residential/"Fenceline" Concentrations Instantaneous Conc. mg/m3	Puddle Conc. mg/m3	TWA mg/m3	On-Site Worker/Building 111 Concentrations Instantaneous Conc. mg/m3	Puddle Conc. mg/m3	TWA mg/m3
1,1 Dichloroethene	1.31E+00	2.02E-03	2.12E-03	2.38E-01	8.73E-04	8.91E-04
1,2 Dichloropropane	4.48E+00	1.53E-03	1.88E-03	8.09E-01	6.47E-04	7.09E-04
Aldrin	4.18E+00	6.72E-08	3.23E-04	7.46E-01	2.84E-08	5.76E-05
Chlorobenzene	4.62E-01	3.88E-05	7.44E-05	8.32E-02	1.66E-05	2.30E-05
Chloroform	1.41E+00	1.80E-03	1.91E-03	2.58E-01	7.79E-04	7.99E-04
Ethyl Benzene	6.72E-01	4.25E-05	9.43E-05	1.21E-01	1.82E-05	2.75E-05
Methylene Chloride	4.52E+01	1.04E-01	1.07E-01	8.27E+00	4.17E-02	4.23E-02
Tetrachloroethene	6.38E-01	1.09E-04	1.58E-04	1.11E-01	4.62E-05	5.48E-05
Toluene	1.77E-01	2.82E-05	4.19E-05	3.20E-02	1.20E-05	1.45E-05
Trichloroethene	4.45E-01	2.36E-04	2.70E-04	8.04E-02	1.02E-04	1.08E-04

Average Concentrations mg/m3

Chemical	Residential/"Fenceline" Concentrations Instantaneous Conc. mg/m3	Puddle Conc. mg/m3	TWA mg/m3	On-Site Worker/Building 111 Concentrations Instantaneous Conc. mg/m3	Puddle Conc. mg/m3	TWA mg/m3
1,1 Dichloroethene	9.11E-01	1.41E-03	1.48E-03	1.66E-01	6.07E-04	6.20E-04
1,2 Dichloropropane	2.24E+00	7.63E-04	9.36E-04	4.04E-01	3.24E-04	3.55E-04
Aldrin	1.60E+00	2.57E-08	1.23E-04	2.86E-01	1.09E-08	2.21E-05
Chlorobenzene	1.63E-01	1.37E-05	2.63E-05	2.94E-02	5.87E-06	8.14E-06
Chloroform	1.04E+00	1.33E-03	1.41E-03	1.90E-01	5.73E-04	5.88E-04
Ethyl Benzene	3.36E-01	2.12E-05	4.71E-05	6.07E-02	9.10E-06	1.38E-05
Methylene Chloride	1.03E+01	2.37E-02	2.45E-02	1.88E+00	9.47E-03	9.61E-03
Tetrachloroethene	5.74E-01	9.78E-05	1.42E-04	9.96E-02	4.16E-05	4.93E-05
Toluene	9.82E-02	1.57E-05	2.33E-05	1.78E-02	6.68E-06	8.05E-06
Trichloroethene	2.78E-01	1.47E-04	1.68E-04	5.02E-02	6.36E-05	6.75E-05

TABLE 4-3

RMA DAY STORAGE TANK
MODELED AIR CONCENTRATIONS

RME Concentrations ug/m3

Chemical	Residential/"Fenceline" Concentrations Instantaneous Conc. mg/m3	Puddle Conc. mg/m3	TWA mg/m3	On-Site Worker/Building 111 Concentrations Instantaneous Conc. mg/m3	Puddle Conc. mg/m3	TWA mg/m3
1,1 Dichloroethene	1.39E-02	3.37E-05	5.68E-05	2.50E-03	1.43E-05	1.84E-05
1,2 Dichloropropane	4.85E-02	2.54E-05	1.06E-04	8.55E-03	1.06E-05	2.48E-05
Aldrin	4.33E-02	1.09E-09	7.22E-05	7.76E-03	4.78E-10	1.29E-05
Chlorobenzene	4.85E-03	6.47E-07	8.73E-06	8.78E-04	2.73E-07	1.74E-06
Chloroform	1.48E-02	2.97E-05	5.43E-05	2.68E-03	1.27E-05	1.71E-05
Ethyl Benzene	6.94E-03	6.94E-07	1.23E-05	1.26E-03	2.95E-07	2.39E-06
Methylene Chloride	4.76E-01	1.67E-03	2.46E-03	8.52E-02	7.30E-04	8.71E-04
Tetrachloroethene	6.45E-03	1.77E-06	1.25E-05	1.15E-03	7.47E-07	2.66E-06
Toluene	1.81E-03	4.89E-07	3.50E-06	3.27E-04	2.03E-07	7.48E-07
Trichloroethene	4.66E-03	3.89E-06	1.17E-05	8.57E-04	1.65E-06	3.08E-06

Average Concentrations ug/m3

Chemical	Residential/"Fenceline" Concentrations Instantaneous Conc. mg/m3	Puddle Conc. mg/m3	TWA mg/m3	On-Site Worker/Building 111 Concentrations Instantaneous Conc. mg/m3	Puddle Conc. mg/m3	TWA mg/m3
1,1 Dichloroethene	9.66E-03	2.35E-05	3.96E-05	1.74E-03	9.94E-06	1.28E-05
1,2 Dichloropropane	2.43E-02	1.27E-05	5.32E-05	4.28E-03	5.31E-06	1.24E-05
Aldrin	1.66E-02	4.17E-10	2.77E-05	2.97E-03	1.83E-10	4.95E-06
Chlorobenzene	1.71E-03	2.28E-07	3.08E-06	3.10E-04	9.63E-08	6.13E-07
Chloroform	1.09E-02	2.19E-05	4.00E-05	1.97E-03	9.31E-06	1.26E-05
Ethyl Benzene	3.47E-03	3.47E-07	6.13E-06	6.29E-04	1.47E-07	1.20E-06
Methylene Chloride	1.08E-01	3.79E-04	5.58E-04	1.93E-02	1.66E-04	1.98E-04
Tetrachloroethene	5.80E-03	1.59E-06	1.13E-05	1.04E-03	6.72E-07	2.40E-06
Toluene	1.00E-03	2.71E-07	1.94E-06	1.82E-04	1.13E-07	4.16E-07
Trichloroethene	2.91E-03	2.43E-06	7.28E-06	5.63E-04	1.03E-06	1.97E-06

TABLE 4-4

LARGE STORAGE TANK FAILURE
INTAKE ASSUMPTIONS FOR VOC INHALATION
HYPOTHETICAL RESIDENTS

$$\text{Intake Factor} = \frac{IR \times ET \times EF \times ED}{BW \times AT}$$

		Average		Reasonable Maximum	
		Child	Adult	Child	Adult
IR =	Inhalation rate, m^3/hour[1]	0.83	0.83	1.25	1.25
ET =	Exposure time, hours/day[2]	16	16	24	24
EF =	Exposure frequency, days/year[3]	9	9	9	9
ED =	Exposure duration, years	1	1	1	1
BW =	Body weight, kg	25[4]	70	25[4]	70
AT =	Averaging time, days[5]				
	Noncarcinogens	9	9	9	9
	Carcinogens	25,550	25,550	25,550	25,550

(1) Based on a rate of 20 and 30 m^3/day for average and RME scenarios, respectively (EPA 1989a).
(2) Average value assumes an 8 hour work or school day; RME value assumes residents are home all day for the full exposure frequency.
(3) Assuming residents are at home for the full duration of the cleanup of 9 days.
(4) Based on average body weight for a child 6 to 9 years (EPA 1989b)
(5) Nine days/year for noncarcinogens; 70 years x 365 days/year for carcinogens.

TABLE 4-5

LARGE STORAGE TANK FAILURE
INTAKE ASSUMPTIONS FOR INHALATION OF VOCS
ON-SITE WORKER

$$\text{Intake Factor} = \frac{IR \times ET \times EF \times ED}{BW \times AT}$$

Parameter	Average	Reasonable Maximum
IR: Inhalation rate, m^3/hour[1]	0.83	0.83
ET: Exposure time, hours/day[2]	8	8
EF: Exposure frequency, days/year[3]	7	7
ED: Exposure duration, years	1	1
BW: Body weight, kg	70	70
AT: Averaging time, days[4]		
Noncarcinogens	7	7
Carcinogens	25,550	25,550

(1) EPA 1989 EFH.

(2) Both the average and reasonable maximum exposure times are conservative estimates of the amount of time a worker would spend on the site each day.

(3) RME and average exposure frequencies of 7 days/year are based on the nine days necessary for cleanup, with one weekend subtracted.

(4) Seven days/year for noncarcinogens; 70 years x 365 days/year for carcinogens.

TABLE 4-6

DAY TANK FAILURE
INTAKE ASSUMPTIONS FOR VOC INHALATION
HYPOTHETICAL RESIDENTS

$$\text{Intake Factor} = \frac{IR \times ET \times EF \times ED}{BW \times AT}$$

		Average		Reasonable Maximum	
		Child	Adult	Child	Adult
IR =	Inhalation rate, m^3/hour[1]	0.83	0.83	1.25	1.25
ET =	Exposure time, hours/day	10	10	10	10
EF =	Exposure frequency, days/year[2]	1	1	1	1
ED =	Exposure duration, years	1	1	1	1
BW =	Body weight, kg	25[4]	70	25[4]	70
AT =	Averaging time, days[6]				
	Noncarcinogens	1	1	1	1
	Carcinogens	25,550	25,550	25,550	25,550

(1) Based on a rate of 20 and 30 m^3/day for average and RME scenarios, respectively (EPA 1989a).
(2) Only one day/year based on the estimated cleanup time.
(4) Based on average body weight for a child 6 to 9 years old (EPA 1989b)
(5) One day/year for noncarcinogens; 70 years x 365 days/year for carcinogens.

TABLE 4-7

DAY TANK FAILURE
INTAKE ASSUMPTIONS FOR INHALATION OF VOCS
ON-SITE WORKER

$$\text{Intake Factor} = \frac{IR \times ET \times EF \times ED}{BW \times AT}$$

Parameter	Average	Reasonable Maximum
IR: Inhalation rate, m^3/hour[1]	0.83	0.83
ET: Exposure time, hours/day[2]	8	8
EF: Exposure frequency, days/year[3]	1	1
ED: Exposure duration, years	1	1
BW: Body weight, kg	70	70
AT: Averaging time, days[4]		
Noncarcinogens	1	1
Carcinogens	25,550	25,550

(1) EPA 1989a.

(2) Both the average and reasonable maximum exposure times are conservative estimates of the time a worker would spend on the site each day.

(3) RME and average exposure frequencies of one day/year are based on the estimated time necessary for cleanup.

(4) One day/year for noncarcinogens; 70 years x 365 days/year for carcinogens.

5.0
TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to evaluate the toxicity of source-related chemicals of concern and to estimate the dose-response relationship for each of these chemicals. The list of chemicals of concern appears in Section 4.1. The information obtained in the toxicity assessment is combined with estimated contaminant intakes calculated as part of the exposure assessment to estimate the potential excess lifetime cancer risks and potential noncarcinogenic health hazards (EPA 1989b).

Noncarcinogenic responses are generally characterized by a threshold: a certain minimum intake of substance below which adverse effects will not occur. Above the threshold, protective mechanisms of the organism may be overwhelmed and effects may occur. Carcinogenic responses are assumed to have no threshold. This assumption means that there is some finite cancer risk no matter how small the dose; the smaller the dose, the smaller the risk of cancer (EPA 1989b).

The two principal indexes of toxicity are the Reference Dose (RfD) and Slope Factor (SF). These values are derived by the EPA for the most commonly occurring and the most toxic chemicals generally associated with chemical releases to the environment. An RfD is an intake or contaminant dose per unit of body weight per day that is unlikely to result in toxic effects to human populations, including sensitive subgroups (e.g., the very young or old). The RfD allows for the existence of a threshold and is used for the assessment of potential noncarcinogenic effects (EPA 1989b).

Chemicals are classified by EPA according to a weight-of-evidence classification. This describes the likelihood that the contaminant causes cancer in humans. For potential carcinogens, the SF is used to estimate the upper bound of the dose-response curve, and is used to estimate the probability of an individual developing cancer as a result of a specific exposure. Table 5-1 presents the EPA weight-of-evidence classification system for carcinogenicity. Slope factors are based on experimental animal data and epidemiological studies when available. A linear nonthreshold mathematical model for

low-dose extrapolation, the linearized multistage model, is used to calculate numerical slope factor value.

The RfD and SF values used in this risk assessment were obtained from the following sources:

- EPA's Integrated Risk Information System (IRIS) (EPA 1992d) on-line database system
- EPA's Health Effects Assessment Summary Tables (EPA 1992e)
- EPA's Health Effects Assessment Summary Tables (EPA 1991b)

Available RfDs and SFs for each contaminant of concern are presented in Table 5-2.

5.1 TOXICITY ASSESSMENT FOR NONCARCINOGENIC EFFECTS

Substances that produce noncarcinogenic effects are generally thought to have a threshold dose below which there are no observable adverse health effects. In developing a toxicity value for noncarcinogenic effects, the approach is to identify this threshold dose or No Observed Adverse Effect Level (NOAEL) through studies with experimental animals or from epidemiological studies. A NOAEL is an experimentally (or epidemiologically) determined highest dose (for a given study) at which there was no statistically or biologically significant effect of concern, often called the "critical toxic effect." For certain substances, only a Lowest Observed Adverse Effect Level (LOAEL) has been determined. This is the lowest dose of a substance that produces either a statistically or biologically significant indication of the critical toxic effect. The NOAEL or the LOAEL may be used to calculate the RfD for a particular contaminant (EPA 1988b).

RfDs are calculated by dividing the NOAEL (or LOAEL) by uncertainty factors, which can range from 10 to 10,000. For example, uncertainties include variations in the sensitivity of individuals within a population and the extrapolation of data from experimental animals to humans. The RfD is expressed in units of milligrams of contaminant per kilogram of body weight per day (mg/kg-day) for oral exposure. Reference Air Concentrations (RfCs) expressed in milligrams of contaminant per cubic

meter of air (mg/m^3) may be available to evaluate inhalation exposure. A body weight of 70 kg and a respiration rate of 20 m^3/day are used to convert the RfC to a dose (mg/kg-day). The methodology for deriving RfDs is more fully described in the EPA's current human health risk assessment guidance (EPA 1989b).

The majority of toxicological knowledge of chemicals comes from experiments on laboratory animals. Experimental animal data have historically been relied upon by regulatory agencies and other expert groups to assess the hazards of human contaminant exposures. Although this reliance has been generally supported by empirical observations, there are known interspecies differences in contaminant absorption, metabolism, excretion, and toxic responses. There are also uncertainties concerning the relevance of animal studies using exposure routes that differ from the human exposure routes under consideration. Additionally, the extrapolation of results of short-term or subchronic animal studies to long-term exposures in humans has inherent uncertainty (EPA 1988b).

Despite the limitations of experimental animal data, such information is essential for contaminant toxicity assessment, especially in the absence of human epidemiological evidence. The uncertainty factors used in the derivation of RfDs are intended to compensate for data limitations and any synergistic effects. Synergistic effects may occur when exposure to a combination of chemicals has a greater than additive effect. Although not typically considered, antagonistic effects can also occur when chemicals of similar chemical orientation (size, molecular weight, etc.) have a lesser effect than either contaminant alone. The RfD approach is conservative by design and is meant to derive protective RfD values (EPA 1989b).

The EPA has developed various types of RfDs depending on the exposure route (ingestion or inhalation), the critical effect, and the length of exposure being evaluated (chronic or subchronic). The EPA bases the RfD on the most sensitive animal species tested (i.e., the species that experiences adverse effects at the lowest dose).

The EPA defines a chronic RfD as an estimate of a daily exposure level for the human population that is unlikely to result in deleterious effects during a lifetime (70 years, according to EPA guidance). A chronic RfD is used to evaluate the potential

noncarcinogenic hazards associated with long-term contaminant exposures (7 years to a lifetime). In this report, chronic RfDs were used when necessary to derive subchronic RfDs.

Subchronic RfDs have been developed to characterize potential noncarcinogenic hazards associated with short-term contaminant exposures. The EPA defines subchronic exposure as periods ranging from two weeks to seven years (EPA 1989b). Subchronic RfDs tend to be higher (greater), generally by one order of magnitude, than chronic RfDs because of the shorter exposure duration.

Although EPA has defined subchronic exposures as being two weeks to seven years, this time frame is a guideline only (EPA 1989b) and in no way reflects clearly delineated exposure periods. In this risk assessment, all exposures are less than 2 weeks, however; subchronic RfDs were used when available. The chronic RfD for toluene was adopted as the subchronic RfD for the inhalation route (EPA 1992e). Because the uncertainty factor was unknown for methylene chloride, the chronic RfD was used as a surrogate for the subchronic RfD for the inhalation route. This is a conservative approach. Subchronic RfDs were derived from chronic RfDs for 1,2 dichloropropane and ethylbenzene by multiplying the chronic RfD by its associated uncertainty factor that accounts for extrapolating short-term laboratory exposures to those assumed to be chronic. These uncertainty factors were taken from current IRIS documentation and were only available for these two COCs. Table 5-2 presents RfDs used in this RA.

Current EPA guidance for assessing risk of noncarcinogenic effects for short-term exposures are divided into three exposure durations and are shown in Table 5-3 (EPA 1991c). Because the failure event of the large tank approximates a subchronic exposure (9 days), subchronic RfDs were used as the short-term toxicity values in order to quantify noncarcinogenic health risks. These toxicity values were used, despite the exposure duration falling somewhat short of the EPA time-frame guidelines. Because subchronic RfDs are set at levels considered protective of developmental effects as well as for other noncarcinogenic effects (EPA 1991c), this approach is conservative.

In addition to subchronic RfDs, other toxicity values are available for assessing risk of noncarcinogenic effects. Two of these toxicity values are Acute Inhalation Criteria

(AIC) and Minimal Risk Levels. The AIC are based on noncancer endpoints, are expressed as air concentrations, and are used for assessing risks from single, very short exposures (up to an hour to a few hours) (EPA 1991c). Twenty-four inhalation MRLs have been developed that span a spectrum of durations including acute, intermediate, and chronic. MRLs were developed using an approach consistent with EPA RfD methodology and, unlike the AIC, are adjusted for exposure duration. Unfortunately, few, if any, of the AIC or inhalation MRLs were available for the COCs in this report.

The estimated instantaneous concentrations developed by the ALOHA model are, for comparison purposes, presented with the Threshold Limit Values (TLVs) and chemical concentrations considered Immediately Dangerous to Life and Health (IDLH) in Tables 5-4 and 5-5. The comparison shows that the instantaneous concentrations are well below these values. However, EPA does not consider these values appropriate for evaluating the exposure scenarios considered in this report as the former is concerned with an immediate occupational setting and the latter were determined only for the purpose of respirator selection (EPA 1991c).

5.2 TOXICITY ASSESSMENT FOR CARCINOGENIC EFFECTS

In estimating the risk posed by potential carcinogens, it is the common practice of the EPA to assume that any exposure level is associated with a finite probability, however minute, of producing a carcinogenic response. EPA assumes that a small number of molecular events can evoke changes in a single cell that can lead to uncontrolled cellular proliferation. This mechanism for carcinogenicity is referred to as "non-threshold" since there is theoretically no level of exposure for such a substance that does not pose a small probability of producing a carcinogenic response. There is increasing evidence that this assumption is not correct for several chemicals. That is, it appears that for at least some chemicals there is a threshold below which the contaminant will not increase the risk of cancer. However, because EPA has not incorporated this knowledge into their risk assessment approach, the traditional, non-threshold approach was used for all potential carcinogens in this report.

The slope factors are usually based on the results of animal studies. There is uncertainty whether animal carcinogens are also carcinogenic in humans. While many contaminant substances are carcinogenic in one or more animal species, only a small number of contaminant substances are known to be human carcinogens. The EPA assumes that humans are as sensitive to all animal carcinogens as the most sensitive animal species. This policy decision is designed to prevent underestimating risk and introduces the potential to overestimate carcinogenic risk (EPA 1989b).

A number of mathematical models and procedures have been developed to extrapolate from carcinogenic responses observed at high doses in experimental animals to responses expected at the low doses humans may be exposed to. The EPA uses a conservative mathematical model, the linearized multistage model, for low-dose extrapolation. The EPA further calculates the upper 95th percent confidence limit of the slope of the resulting dose-response curve. This SF value, expressed in units of $(mg/kg\text{-}day)^{-1}$, is used to convert the average daily intake of a contaminant, averaged over a lifetime, to a cancer risk. This represents an estimation of an upper-bound incremental lifetime probability that an individual will develop cancer as a result of exposure to a potential carcinogen. This model provides a conservative estimate of cancer risk at low doses and is likely to overestimate the actual cancer risk. The EPA acknowledges that actual slope factors are likely to be between zero and the estimate provided by the linearized multistage model (EPA 1989b). The weight-of-evidence classifications and slope factors for the chemicals of concern are included in Tables 5-1 and 5-2, respectively.

There is little guidance available for estimating cancer risks from short-term exposures to carcinogens. Typically, for recognized and/or potential carcinogens, excess lifetime cancer risks are obtained by considering the cumulative dose, averaged over a lifetime (EPA 1989b; EPA 1986). EPA recommends this approach always be used to estimate carcinogenic risks (EPA 1989b). Although other methods have been developed recently for assessing carcinogenic risks due to short-term exposures (EPA 1991c), the standard EPA-recommended methodology described above is used here.

TABLE 5-1

EPA WEIGHT-OF-EVIDENCE CARCINOGENIC CLASSIFICATION OF CONTAMINANTS

Group	Description	Description of Evidence
A	Human carcinogen	Sufficient evidence from epidemiologic studies to support a causal association between exposure and cancer
B1 or B2	Probable human carcinogen	B1 indicates that limited human data are available from epidemiologic studies. B2 indicates sufficient evidence in animals and inadequate or no evidence in humans of carcinogenicity.
C	Possible human carcinogen	Limited evidence of carcinogenicity in animals
D	Not classifiable as to human carcinogenicity	Inadequate evidence of carcinogenicity in animals
E	No evidence of carcinogenicity in humans	No evidence of carcinogenicity in at least two adequate animal tests or in both epidemiologic and animal studies.

Source: EPA 1989b

TABLE 5-2

SUBCHRONIC RfDs AND SLOPE FACTORS* FOR CHEMICALS OF CONCERN

		Toxicity		Carcinogenicity		
Compound	Exposure Route	Subchronic RfD (mg/kg-day)	Uncertainty[a] Factor	Slope Factor (mg/kg-day)[-1]	EPA Weight of Evidence Category	Source
1,1-Dichloroethene	Inhalation	Pending[b] ND	NA NA	1.75E-01[d]	C	IRIS 2/93 HEAST FY92
1,2-Dichloropropane	Inhalation	3.4E-03[e]	3	ND ND	ND	IRIS 2/93 HEAST FY92
Aldrin	Inhalation	ND ND	NA NA	1.7E+01[d]	B2	IRIS 2/93 HEAST FY92
Chlorobenzene	Inhalation	Pending[b] 5.7E-02[e]	NA NA	ND ND	D	IRIS 2/93 HEAST FY92
Chloroform	Inhalation	Pending[b] Pending[b]	NA NA	8.1E-02[d,f]	B2	IRIS 2/93 HEAST FY92
Ethylbenzene	Inhalation	2.9E+00	10	ND ND	D	IRIS 2/93 HEAST FY92
Methylene Chloride	Inhalation	Pending[b] 8.6E-01[b,c]	NA NA	1.64E-03[d]	B2	IRIS 2/93 HEAST FY92
Tetrachloroethene	Inhalation	ND ND ND	NA NA NA	ND ND 1.82E-03	B2	IRIS 2/93 HEAST FY92 HEAST FY91
Toluene	Inhalation	1.1E-01	NA	ND ND	D	IRIS 2/93 HEAST FY92
Trichloroethene	Inhalation	WD ND ND	NA NA NA	WD ND 6.2E-03	B2	IRIS 2/93 HEAST FY92 HEAST FY91

*The source of the toxicity values reported here follow the hierarchy put forth by EPA. That is, the Integrated Risk Information System (IRIS) and the Health Effects Assessment Summary Tables (HEAST) are to be used as first and second choices, respectively, for toxicity data in risk assessments. It should be noted that data listed in HEAST are provisional information only.

[a] Obtained from IRIS. This uncertainty factor converts a chronic exposure to a subchronic exposure when multiplied by the RfD_i.

[b] Under review by EPA Work Group.

[c] Converted from unit risk to chronic RfD_i then converted to subchronic RfD_i using chemical specific uncertainty factor. Toluene represents a chronic RfD_i adopted as a subchronic RfD_i (EPA 1992 HEAST Suppl. 2). Methylene chloride was only converted from a chronic unit risk; the uncertainty factor was unknown. Therefore, this chronic RfD_i was used as a surrogate value for the subchronic RfD_i for methylene chloride.

[d] Converted from unit risk to slope factor by method described in HEAST.

[e] The subchronic inhalation RfC value was derived from methodology not current with the interim inhalation methodology used by the RfD/RfC EPA Work Group.

[f] Route-to-route extrapolation; intrinsic to study that developed slope factor unit risk.

ND = Not Determined by EPA
NA = Not Applicable
WD = Withdrawn from IRIS as of 2/93

TABLE 5-3

SHORT-TERM EXPOSURES OF POTENTIAL CONCERN

- **Single Exposure Event.** The majority of chemicals are capable of producing an adverse health effect after a single exposure event, depending on the intensity of exposure. For developmental toxicants, irritants, and neurological poisons, a single, low level exposure event can result in effects after minutes, hours, or a day.
- **Very Short-term Exposure.** For some acute toxicants, multiple exposures over several days could result in an adverse effect. For these chemicals, the exposure is assessed over days or weeks (up to two weeks).
- **Short-term (Subchronic) Exposure.** Exposure lasting anywhere from two weeks to seven years to low concentrations of a chemical can also produce adverse effects; this exposure is assessed by averaging it over the specific duration.

Source: EPA 1991c

TABLE 5-4

RMA LARGE STORAGE TANK
RME CONCENTRATIONS

Chemical	Residential/Fenceline Instantaneous Concentration		On-Site Worker/Building 111 Instantaneous Concentration		IDLH	TLV STEL
	ppm	mg/m^3	ppm	mg/m^3		
1,1-Dichloroethene	.330	1.31E+00	.060	2.38E-01	--	20 ppm[b]; 80 mg/m^3
1,2-Dichloropropane	.970	4.48E+00	.175	8.09E-01	(Ca) 2,000 ppm[a]	110 ppm[a,b,c]; 510 mg/m^3
Aldrin	.280	4.18E+00	.050	7.46E-01	(Ca) 100 mg/m^3[3a]	---
Chlorobenzene	.100	4.62E-01	.018	8.32E-02	2,400 ppm[a]	---
Chloroform	.290	1.41E+00	.053	2.58E-01	(Ca) 1,000 ppm[a]	2 ppm[a,c]; 9.78 mg/m^3
Ethylbenzene	.155	6.72E-01	.020	1.21E-01	2,000 ppm[a]	125 ppm[a,b,c]; 545 mg/m^3
Methylene Chloride	13.0	4.52E+01	2.38	8.27E+00	(Ca) 5,000 ppm[a]	500 ppm[b]; 1,740 mg/m^3
Tetrachloroethene	.094	6.38E-01	.016	1.11E-01	(Ca) 500 ppm[a]	200 ppm[b]; 1,340 mg/m^3
Toluene	.047	1.77E-01	.009	3.20E-02	2,000 ppm[a]	150 ppm[a,b,c]; 560 mg/m^3
Trichloroethene	.083	4.45E-01	.015	8.04E-02	(Ca) 1,000 ppm[a]	200 ppm[a,b,c]; 1,080 mg/m^3

IDLH = Immediately Dangerous to Life and Health
TLV STEL = Threshold Limit Value, Short Term Exposure Limit
Ca = Carcinogenic

[a] NIOSH Pocket Guide to Chemical Hazards, U.S. Dept. of Health & Human Services. June 1990.
[b] Threshold Limit Values and Biological Exposure Indices for 1986-1987, American Conference of Governmental Industrial Hygienists.
[c] Guide to Occupational Exposure Values - 1992, Compiled by the American Conference of Governmental Industrial Hygienists.

TABLE 5-5

RMA DAY STORAGE TANK
RME CONCENTRATIONS

Chemical	Fenceline Concentrations Instantaneous Concentration		Building 111 Concentrations Instantaneous Concentration		IDLH	TLV STEL
	ppm	mg/m^3	ppm	mg/m^3		
1,1-Dichloroethene	.0035	1.39E-02	.0006	2.50E-03	--	20 ppm[b]; 80 mg/m^3
1,2-Dichloropropane	.0105	4.85E-02	.0018	8.55E-03	(Ca) 2,000 ppm[a]	110 ppm[a,b,c]; 510 mg/m^3
Aldrin	.0029	4.33E-02	.0005	7.76E-03	(Ca) 100 mg/m^3[3a]	---
Chlorobenzene	.0011	4.85E-03	.0002	8.78E-04	2,400 ppm[a]	---
Chloroform	.0031	1.48E-02	.0006	2.68E-03	(Ca) 1,000 ppm[a]	2 ppm[a,c]; 9.78 mg/m^3
Ethylbenzene	.0016	6.94E-03	.0003	1.26E-03	2,000 ppm[a]	125 ppm[a,b,c]; 545 mg/m^3
Methylene Chloride	.1370	4.76E-01	.0245	8.52E-02	(Ca) 5,000 ppm[a]	500 ppm[b]; 1,740 mg/m^3
Tetrachloroethene	.0009	6.45E-03	.0002	1.15E-03	(Ca) 500 ppm[a]	200 ppm[b]; 1,340 mg/m^3
Toluene	.0005	1.81E-03	.00009	3.27E-04	2,000 ppm[a]	150 ppm[a,b,c]; 560 mg/m^3
Trichloroethene	.0009	4.66E-03	.0002	8.57E-04	(Ca) 1,000 ppm[a]	200 ppm[a,b,c]; 1,080 mg/m^3

IDLH = Immediately Dangerous to Life and Health
TLV STEL = Threshold Limit Value, Short Term Exposure Limit
Ca = Carcinogenic

[a] NIOSH Pocket Guide to Chemical Hazards, U.S. Dept. of Health & Human Services. June 1990.
[b] Threshold Limit Values and Biological Exposure Indices for 1986-1987, American Conference of Governmental Industrial Hygienists.
[c] Guide to Occupational Exposure Values - 1992, Compiled by the American Conference of Governmental Industrial Hygienists.

6.0
RISK CHARACTERIZATION

Risk characterization is the final step of the risk assessment process. In this step, the toxicity factors (RfDs and cancer slope factors) for the chemicals of concern are applied in conjunction with estimated chemical intakes to predict noncarcinogenic and carcinogenic health risks to exposed individuals.

The potential for noncarcinogenic effects is characterized by comparing estimated chemical intakes with chemical-specific RfDs. In this report, subchronic (2 weeks to 7 years) hazard indexes are the appropriate indicator of potential non-cancer adverse effects for all exposures. Potential carcinogenic effects are characterized in terms of the incremental or excess probability that an individual will develop cancer in his or her lifetime due to the modeled exposure. Excess cancer risk is estimated from the projected lifetime intakes and the cancer slope factor, which represents an upper-bound estimate of the dose-response relationship.

Potential health risks associated with the estimated chemical exposures are presented in Section 6.2. Uncertainties inherent in the risk assessment process are presented in Section 7.0.

6.1 METHODOLOGY FOR QUANTITATIVE RISK ESTIMATION

6.1.1 Hazard Index For Noncarcinogenic Effects

The potential for adverse noncarcinogenic effects resulting from exposure to a chemical of concern is evaluated by comparing the average daily intake of the chemical (expressed as mg/kg-day)) to a reference dose (expressed as mg/kg-day). The resulting ratio is called a hazard quotient (EPA 1989b). It is derived in the following manner:

$$\text{Noncancer Hazard Quotient} = \frac{\text{Chemical Intake (mg/kg-day)}}{\text{RfD (mg/kg-day)}}$$

Use of the RfD assumes a level of intake (the RfD) below which it is unlikely that even sensitive individuals will experience adverse health effects over a lifetime of exposure. If the average daily intake exceeds the RfD (that is, if the hazard quotient exceeds 1.0), there may be cause for concern for potential noncancer effects. It should be noted, however, that the level of concern does not increase linearly as the RfD is approached or exceeded. This is because all RfDs are not equally accurate and are not based on the same severity of toxic effects. Thus, the slopes of the dose-response curve in excess of the RfD can vary widely depending on the substance. Furthermore, it must be emphasized that the hazard quotient incorporates uncertainty factors, and it does not represent a statistical probability of an effect occurring.

To assess the overall potential for adverse health effects posed by exposure to multiple chemicals, the hazard quotients for each chemical of concern associated with a given exposure pathway are summed. The resulting sum is referred to by EPA as the hazard index (H.I.). The H.I. approach assumes that multiple subthreshold exposures to several chemicals could result in an adverse health effect. The H.I. is expressed as follows:

$$H.I. = E_1/RfD_1 + E_2/RFD_2 \ldots + E_i/RfD_i$$

where

E_i = Exposure level (or intake) for toxicant i
RfD_i = Reference dose for toxicant i

For multiple chemical exposures, the total H.I. might exceed 1.0 even if no single chemical intake exceeds its RfD. If the total H.I. is less than 1.0, cumulative exposures to the chemicals of concern at the site is judged unlikely to result in an adverse effect. If the sum is greater than 1.0, a more detailed and critical evaluation of the risks including consideration of specific target organs affected and mechanisms of toxic action of the chemicals of concern is required to ascertain if the cumulative exposure would be likely to harm exposed individuals.

The assumption of additive effects reflected in the cumulative H.I. is most properly applied to substances that induce the same effect by the same mechanism (EPA 1989b).

Consequently, application of the equation to a mixture of substances that are not expected to induce the same type of effects could overestimate the potential for adverse health effects. The H.I. provides a rough measure of potential toxicity, but it is conservative and dependent on the quality of the experimental evidence. Since the H.I. does not define dose-response relationships, its numerical value cannot be construed as a direct estimate of risk (EPA 1989b), but only as a level of concern.

6.1.2 Risk for Carcinogenic Effects

Carcinogenic risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of site-related exposure to a potential carcinogen. The numerical estimate of excess lifetime cancer risk is calculated by multiplying the chemical intake averaged over a lifetime by the cancer SF as follows:

$$\text{Risk} = \text{Chemical Intake (mg/kg-day)} \times \text{SF (mg/kg-day)}^{-1}$$

The EPA slope factors are upper 95th percentile confidence limits of the probability of response per unit intake of chemical over a lifetime. EPA states that carcinogenic risks estimated using this approach are upper-bound estimates. This means that the actual risk is likely to be less than the predicted risk (EPA 1989b).

EPA guidance to evaluate cancer risk from simultaneous exposure to several carcinogens assumes that incremental cancer risks are additive. The concept that cancer risks are additive is based on a number of assumptions. If these assumptions are incorrect, over- or under-estimation of the actual risk could result (EPA 1989b). The total cancer risk is estimated by summing the risks estimated for each chemical of concern and for each pathway.

EPA policy must be considered in order to interpret the significance of the cancer risk estimates. Current EPA guidance states that where the cumulative site risk to an individual based on reasonable maximum exposure is less than 1×10^{-4}, action is generally not warranted (EPA 1991d). A carcinogenic risk level of 1×10^{-4} was therefore used as the benchmark risk level for evaluation of potential exposures in this preliminary risk assessment.

6.1.3 Risk Quantification

The potential for noncarcinogenic health effects due to a chemical of concern, expressed as a hazard quotient, is calculated using the following format:

(1)	(2)	(3) (1) x (2)	(4)	(5) (3) ÷ (4)
Chemical Concentration (mg/y)	Intake Factor (y/kg-day)	Daily Intake (mg/kg-day)	RfD (mg/kg-day)	Hazard Quotient

Where y = specific amount (weight or volume) of medium in which the chemical is measured; the units of which are medium dependent. For example, a chemical in soil may be reported in mg/kg and in water in mg/liter.

Average or RME concentrations of each noncarcinogenic chemical of concern (column 1) are multiplied by the pathway-specific intake factors (column 2) to yield an average or RME daily intake (column 3). The daily intake is then divided by the RfD (column 4) to obtain the hazard quotient for average or reasonable maximum exposures.

Individual hazard quotients are summed for all chemicals in an exposure pathway to provide a pathway-specific hazard index. Pathway-specific indexes are summed to obtain the total hazard index for all exposure pathways applicable to a given receptor.

Hazard indexes of noncarcinogenic risks for each receptor and pathway are summarized in Section 6.2. Hazard indexes are calculated for subchronic exposures for receptors at each of the exposure locations. Risk calculations are shown in Appendix B.

The potential for carcinogenic effects, expressed as excess cancer risk, are calculated in a similar manner:

(1)	(2)	(3) (1) x (2)	(4)	(5) (3) x (4)
Chemical Concentration (mg/y)	Lifetime Intake Factor (y/kg-day)	Lifetime Daily Intake (mg/kg-day)	Slope Factor $(mg/kg\text{-}day)^{-1}$	Excess Cancer Risk

Where y = specific amount (weight or volume) of a medium in which the chemical is measured; the units of which are medium dependent.

Concentrations of each potential carcinogen (column 1) are multiplied by pathway-specific intake factors (column 2) to yield a lifetime daily intake (column 3). The daily intake is then multiplied by the slope factor to obtain a numerical estimate of excess cancer risk.

The cancer risks are then summed for all of the chemicals evaluated for each exposure pathway. To determine the total carcinogenic risk to a given receptor, the cancer risks for each exposure pathway are summed. Cancer risk calculations are shown in Appendix B.

6.2 RISK CHARACTERIZATION

Results for the human health risk assessment indicate that no noncarcinogenic hazard indexes exceeded the EPA level of concern (1.0) for any of the exposure scenarios evaluated for either failure event using average or reasonable maximum exposure assumptions (Tables 6-1 and 6-2).

For the failure event of the day tank, the greatest hazard index for a receptor for average exposure assumptions is 5.43×10^{-3} and represents the child residential receptor. The greatest hazard index for RME assumptions for the day tank is also for the child receptor (1.71×10^{-2}). For the failure event of the large tank, the greatest total hazard index for a receptor for average exposure assumptions is 1.62×10^{-1} and represents the child resident receptor. The greatest hazard index for RME assumptions for the large tank failure is 8.15×10^{-1} and also represents the resident child receptor.

No carcinogenic risks estimated exceeded the EPA acceptable risk level of 1×10^{-4} for any exposure pathways evaluated for all receptors considered (Tables 6-1 and 6-2). Toxicity profiles for the chemicals that contribute 99 percent of the risk are contained in Appendix C.

TABLE 6-1

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARDS
RMA LARGE STORAGE TANK FAILURE

Receptor/Pathway	Average Exposure		Reasonable Maximum Exposure	
	Cancer Risk	Subchronic Hazard Index	Cancer Risk	Subchronic Hazard Index
Office Worker -- Inhalation of Air on-site	1.42E-08	1.10E-02	3.30E-08	2.45E-02
Adult Resident -- Inhalation of Air at Fenceline	1.67E-07	5.78E-02	9.35E-07	2.91E-01
Child Resident -- Inhalation of Air at Fenceline	4.69E-07	1.62E-01	2.62E-06	8.15E-01

TABLE 6-2

CARCINOGENIC RISKS AND NONCARCINOGENIC HAZARDS
RMA DAY STORAGE TANK FAILURE

Receptor/Pathway	Average Exposure		Reasonable Maximum Exposure	
	Cancer Risk	Subchronic Hazard Index	Cancer Risk	Subchronic Hazard Index
Office Worker -- Inhalation of Air on-site	3.26E-10	3.69E-04	8.37E-10	7.92E-04
Adult Resident -- Inhalation of Air at Fenceline	2.24E-09	2.06E-03	8.71E-09	6.11E-03
Child Resident -- Inhalation of Air at Fenceline	6.26E-09	5.43E-03	2.44E-08	1.71E-02

7.0
UNCERTAINTIES AND LIMITATIONS

The EPA guidance for risk assessment provides a systematic means for organizing, analyzing, and presenting information on the nature and magnitude of potential risks to public health and the environment posed by chemical exposures. Despite the advanced state of the current methodology, uncertainties and limitations are inherent in the risk assessment process. The quality of available data, incomplete information about existing conditions and future circumstances, as well as other factors discussed below contribute to these uncertainties and limitations. This section discusses the following sources of uncertainties and limitations associated with this risk assessment:

- Data collection and evaluation
- Exposure assessment
- Toxicity assessment
- Risk characterization

7.1 Data Collection and Evaluation

Data used in the Human Health Risk Assessment were collected by various companies. The data collected are subject to uncertainty associated with sampling and analysis.

It was assumed that samples were analyzed using Contract Laboratory Program (CLP) procedures. Sample analysis is subject to uncertainties associated with precision, accuracy, and detection of chemicals at low concentrations, which are generally random errors which may lead to over- or underestimation of risks. These errors are typically of low magnitude (well below an order of magnitude) compared to other sources of uncertainty in the risk assessment.

Some chemicals were listed as "NA" or not analyzed, leading to a possible underestimation of risk. It was assumed that these chemicals listed as not analyzed was based on historical data that showed these chemicals to be absent from the medium being tested.

The arithmetic mean and the maximum concentrations of chemicals measured in Basin F liquid were compiled for use in the risk assessment. Normally, it is assumed that a chemical not detected in a given sample is actually present at one-half of its detection limit, if that chemical was present in any sample in a given medium. This assumption was unnecessary based on data used as explained above. The arithmetic mean concentration was used to evaluate average exposures, an assumption which, compared to the geometric mean, overestimates risk because the arithmetic mean is usually greater. In estimating RME scenario, the maximum concentrations were used, which likely results in overestimation of potential risk as the maximum is also usually greater than the upper confidence limit on the arithmetic mean.

7.2 EXPOSURE ASSESSMENT

The exposure assessment is based on site-specific chemical concentration data and on assumptions concerning receptor behavior leading to exposure (time spent at the exposure point).

The assumption was made that these ambient concentrations remained constant over the entire time required to clean up each spill. This is very conservative and likely overestimates risk. In reality, the air containing contaminants would require time to travel to the receptor. Also, as the spills are removed, the surface area exposed to the atmosphere and, hence, the volatilization decreases.

The exposure assessment relied on assumptions of two specific failure events, each of these having an average and RME exposure scenario. The average case scenarios represent assumptions which are considered central values or realistically conservative estimates for the exposed population. The RME scenarios are developed to provide an upper-bound risk estimate. The RME scenarios are based on combinations of conservative assumptions for all variables related to exposure, and thus are highly likely to overestimate potential risks, possibly by a large amount (one or more orders of magnitude).

Assumptions concerning most of the generic (non-site-specific) variables used in estimating chemical intakes are based on data collected for human populations, and thus

are subject to limited uncertainty. These include variables such as body weights, inhalation rates, and activity patterns. The toxicity of inhaled vapors of volatile and semi-volatile organic chemicals is dependent on bioavailability, or the amount of chemicals absorbed through the alveolar wall and other parts of the lung into the systemic bloodstream. The assumption of 100 percent bioavailability is highly conservative and leads to overestimation of risk.

7.3 TOXICITY ASSESSMENT

In general, the available scientific information is insufficient to provide a thorough understanding of all the potential toxic properties of chemicals to which humans are potentially exposed. Consequently, varying degrees of uncertainty surround the assessment of adverse health effects in exposed populations. Sources of uncertainty related directly to toxicity data include:

- Use of dose-response data from experiments on homogenous, sensitive animal populations to predict effects in heterogenous human populations with a wide range of sensitivities

- Extrapolation of data from: (1) high dose animal studies to low-dose human exposures, (2) LOAELs NOAELs acute to subchronic exposure and (3) one exposure route to another (e.g., from ingestion to inhalation; this was only the case for chloroform and was intrinsic to the study itself. That is, the authors extrapolated their results from a gavage (oral) dose to a delivered dose represented as a concentration in air).

- Use of single-chemical test data that do not account for multiple exposures or synergistic and antagonistic responses

The majority of toxicological knowledge of chemicals comes from experiments with laboratory animals due to the limited studies available for humans.

Although animal experimental data have many limitations, they are widely believed to be a necessary part of toxicity assessment, especially in the absence of human

epidemiological evidence. Use of animal data for human health assessment may under- or overestimate potential human health risk. In order to account for this uncertainty, EPA generally applies safety factors ranging from 10 to 1,000 for the results of studies in sensitive animal populations to develop toxicity values to be used in human health evaluations. This conservative approach is likely to overestimate the potential human health risks.

The model used by EPA to determine slope factors for carcinogens is a linearized multistage model, which provides a conservative estimate of cancer risk at low doses and is likely to overestimate the actual slope factor. EPA acknowledges that the actual slope factor could be as low as zero. Inadequate knowledge of the validity and accuracy of this model, however, increases uncertainty and the tendency to overestimate cancer risks.

"Synergistic" effects are those in which the net effect of the simultaneous action of two or more toxic agents is greater than the sum of their individual effects. "Antagonistic" effects are those in which the net effect of exposure to two or more agents is less than the sum of their individual effects. Data concerning combined effects of complex mixtures of chemicals are generally lacking. In the absence of such data, the approach recommended by EPA for evaluating the health impact of chemical mixtures assumes dose adaptivity. This approach does not account for potential synergism, antagonism, or differences in target organ specificity and mechanism of action. This approach may over- or underestimate the potential human health impact.

7.4 RISK CHARACTERIZATION

Because there are uncertainties in each step of the risk assessment process, these uncertainties are often magnified in the final risk characterization. The final quantitative estimates of risk may be one or several orders of magnitude different from the potential risk associated with a given exposure. In an attempt to minimize the consequences of uncertainty, EPA guidance typically relies on use of conservative estimates of hazards in the absence of strong scientific data. The overall result is that risk estimates presented in this report are much more likely to overestimate the potential risk rather than to underestimate it.

The assessment has been prepared in a manner consistent with that generally used in the consulting community and agency guidance at the time it was prepared. It is likely that risk assessment methods and data identifying and quantifying the toxicity of chemicals will improve with time. This assessment was based on recently collected data using the current available risk assessment methodology.

8.0
SUMMARY OF RESULTS

The approach used in this risk assessment followed EPA guidance for conducting human health risk assessments at Superfund sites (EPA 1989e; EPA 1988b; EPA 1991a,b). The EPA cautions that these documents are intended to provide guidance only and that considerable professional judgment must be exercised in applying the guidance to site-specific human health risk assessments. This risk assessment has incorporated conservative assumptions, in addition to site-specific information, in evaluating potential exposures so that potential health risks would not be underestimated.

The results of this risk assessment indicate that, upon exposure to modeled air concentrations using modeled emissions of COCs from both the day and large storage tanks at Basin F, no noncarcinogenic hazard index exceeded the EPA level of concern of unity (1.0) and no carcinogenic risks estimated exceeded the EPA benchmark risk level of 1×10^{-1} for any exposure scenario evaluated (Tables 6-1 and 6-2).

It should be restated that the RME scenario in this risk assessment used a combination of site-specific data and conservative assumptions that represent the "high-end" exposure and more than likely overestimates the actual total cancer risk given the exposure and behavior patterns assumed for this assessment.

Thus, based on the conservative (protective) approach used in this report, catastrophic failure of a Basin F liquid storage tank would not be expected to present significant cancer or non-cancer risks to on-site office workers and off-site child and adult residents.

9.0
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APPENDIX A
SPILL SCENARIO ASSUMPTIONS AND CALCULATIONS

Subject RMA RISK ASSESSMENT

Project No. 89C114MM

By JD PORTER

Checked By TA Brogan

Task No. 13

File No.

Date 3-10-93

Date 3/11/93

Sheet of

- NEAREST FENCE LINE FROM TK-101, 102, 103 AREA IS 5,650' DIRECTLY NORTH.

- DISTANCE FROM TK-101, 102, 103 AREA TO BUILDING 111 IS 9,700'.

Subject RMA RISK ASSESSMENT

Project No. 89C114MM

By JDPORTER Checked By TAB

Task No. 13

File No.

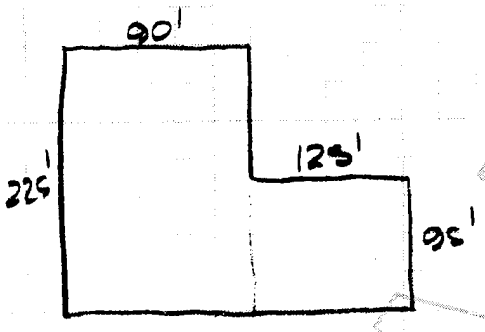
Date 3-10-93 Date 3/11/93

Sheet of

ESTIMATE SURFACE AREA OF LIQUID IN TANK CONTAINMENT AREA IN THE EVENT THAT ONE TANK FULL OF LIQUID WERE TO EMPTY ALL OF ITS CONTENTS

(1) INTIALLY CALCULATE VOLUME IN DRY POND LOCATED IN CONTAINMENT AREA

IGNORE RADIUS FOR QUICK APPROXIMATION



NOMINAL DEPTH =

5185.5

−5180.8

4.7'

VOLUME (CF) = [225×90 + 125×95] × 4.7'

= [20,250 + 11,875] × 4.7'

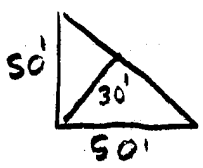
= [32,125] × 4.7

= 150,987 CF ✓

= 150,987 CF × 7.4805 GALS/CF

= 1,129,462 GALS ✓

ADD APPROXIMATE VOLUME FOR RADIUS PORTION



VOLUME = $\frac{1}{2}$ (50×50) × 4.7'

= 1250×4.7 = 5875 CF ✓

= 43,948 GALS ✓

Subject RMA RISK ASSESSMENT

Project No. 89C114MM

By JD PORTER Checked By TAB

Task No. 13

File No.

Date 3-10-93 Date 3/11/93

Sheet of

TOTAL HOLDING VOLUME IN DRY POND =

1,129,462
43,948

1,173,410 GALLONS ✓

TANKS HOLD 1,333,000 GALLONS/EACH

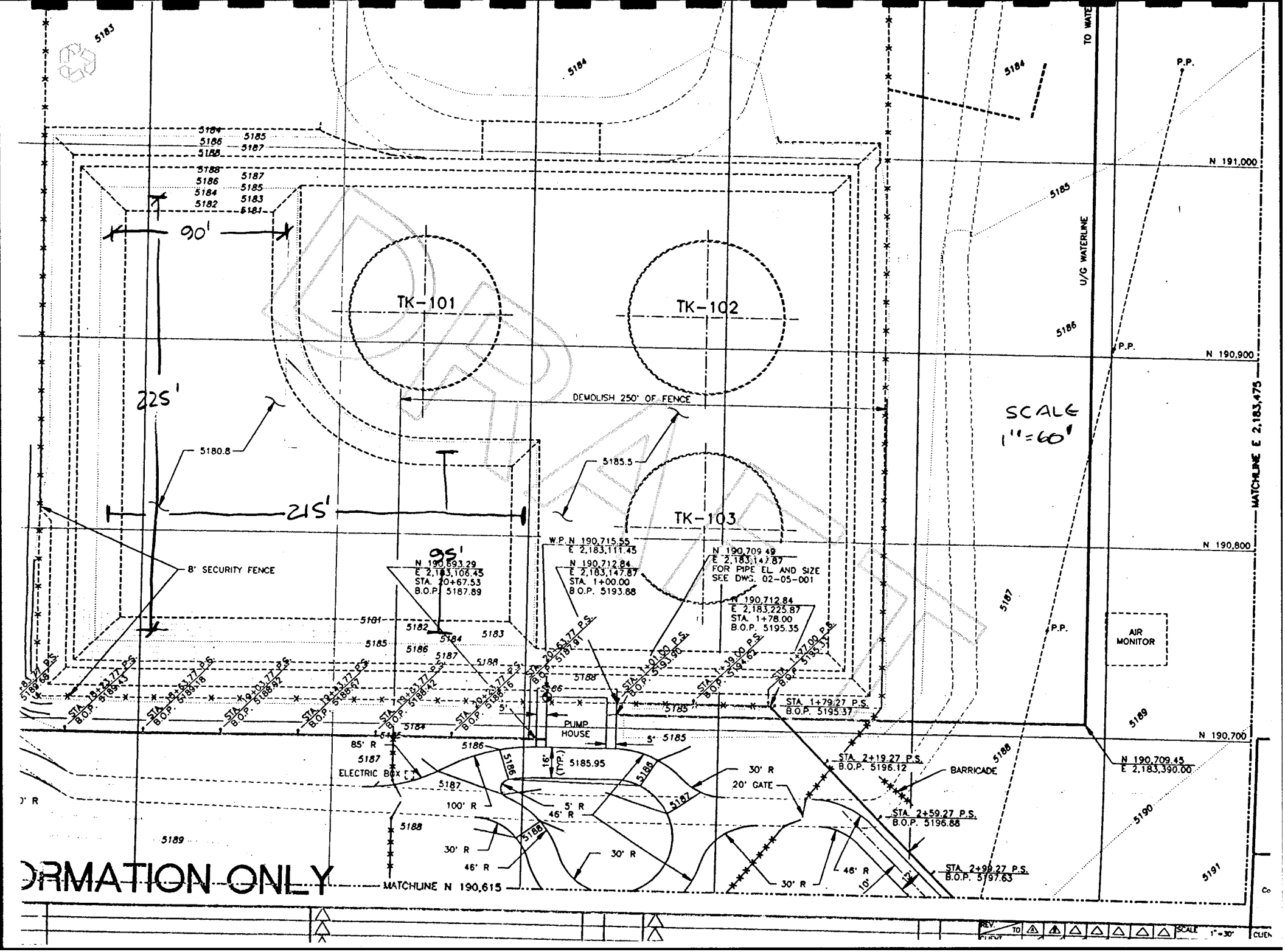
WORST CASE ASSUMPTION WOULD ASSUME ENTIRE CONTAINMENT AREA WOULD BE WET

$A = 380' \times 240' - 3\pi \left(\frac{78.6}{2}\right)^2$

$= 91,200 - 14,556 = 76,644$ SF ← USE ✓

A MORE PRACTICAL ASSUMPTION WOULD BE THE SURFACE OF THE DRY POND PLUS 10% FOR WET SOIL BEFORE LIQUID RUNS INTO DRY POND

$A = [32,125 + 1250] \times 1.1 = 36,713$ SF


TK-101
TK-102
TK-103
90'
225'
215'
95'
5180.8
5185.5
8' SECURITY FENCE
DEMOLISH 250' OF FENCE
SCALE
1"=60'
U/G WATERLINE
MATCHLINE E 2,183,475
MATCHLINE N 190,615
N 191,000
N 190,900
N 190,800
N 190,700
N 190,693.29
E 2,183,106.45
STA. 20+67.53
B.O.P. 5187.89
W.P. N 190,715.55
E 2,183,111.45
N 190,712.84
E 2,183,147.87
STA. 1+00.00
B.O.P. 5193.88
N 190,709.49
E 2,183,147.87
FOR PIPE EL. AND SIZE
SEE DWG. 02-05-001
N 190,712.84
E 2,183,225.87
STA. 1+78.00
B.O.P. 5195.35
N 190,709.45
E 2,183,390.00
STA 1+79.27 P.S.
B.O.P. 5195.37
STA 2+19.27 P.S.
B.O.P. 5196.12
STA 2+59.27 P.S.
B.O.P. 5196.88
STA 2+99.27 P.S.
B.O.P. 5197.63
PUMP HOUSE
ELECTRIC BOX
BARRICADE
20' GATE
AIR MONITOR
5185.95
16' (TYP.)
85' R
100' R
46' R
30' R
5' R
5' 5185
ORMATION ONLY

Subject RMA TANK SPILL RISK ASSESSMENT - STORAGE TANKS

Project No. 8XC114MM

By KS BROGAN

Checked By GD Porter

Task No. 13

File No.

Date 3/15/93

Date 3/15/93

Sheet of

LENGTH OF TIME EXPOSED TO AIR AFTER TANK FAILURE

ASSUME : LIQUID WOULD BE PUMPED / TRANSFERRED TO POND A

SCENARIO 1 - ONE VACUUM TRUCK - 5500 gallons

ASSUME: 1 hr TO LOAD
½ hr TO UNLOAD
¼ hr. DRIVE
} 105 minute cycle

$\frac{5500 \text{ gal}}{105 \text{ min}} = 52 \text{ gpm}$

$\frac{1,333,000 \text{ gallons}}{52 \text{ gpm}} = 25635 \text{ minutes} = 427 \text{ hours} = 18 \text{ days}$

SCENARIO 2 - ONE 50 gpm PUMP and 2-inch LINE TO POND A

$\frac{1333000 \text{ gal}}{50 \text{ gpm}} = 26660 \text{ minutes} = 445 \text{ hours} = 18\frac{1}{2} \text{ days}$

SCENARIO 3 - TWO VACUUM TRUCKS - 5500 gallons EACH IN THE 105 minute CYCLE (SEE SCENARIO 1)

$\frac{2(5500 \text{ gal})}{105 \text{ min}} = 104 \text{ gpm}$

$\frac{1333000 \text{ gal}}{104 \text{ gpm}} = 12817 \text{ minutes} = 214 \text{ hours} = \boxed{9 \text{ days}}$

SCENARIO 4 - 50 gpm PUMP AND TWO VACUUM TRUCKS

$\frac{1333000 \text{ gallons}}{50 + 104 \text{ gpm}} = 8656 \text{ minutes} = 144 \text{ hours} = 6 \text{ days}$

SCENARIO 5 - 50 gpm PUMP AND ONE VACUUM TRUCK

$\frac{1333000 \text{ gal}}{50 + 52 \text{ gpm}} = 13,069 \text{ minutes} = 218 \text{ hours} = 9 \text{ days}$

USE SCENARIO 3

Subject RMA TANK SPILL RISK ASSESSMENT - DAY TANKS Project No. 92C114MM

By KS BROGAN Checked By GD Porter Task No. 13

Date 3/12/93 Date 3/12/93 File No.

Sheet 1 of 1

DAY TANKS (105, 106) VOLUME 14,000 gallons

from Final O&M Manual, Volume I, September 1992, Weston

DIMENSIONS 12FT DIAMETER
17FT HEIGHT

CONTAINMENT AREA CAPACITY 33,770 gallons

4.5 ft high sides

from Final O&M Manual Vol. III, Appendix A, Site H&S Plan, Sept. 1992 Weston

$$(33770 \text{ gallons})\left(\frac{0.1336 \text{ CF}}{\text{gal}}\right) = 4511 \text{ CF}$$

$$\frac{4511 \text{ CF}}{4.5 \text{ FT HEIGHT}} = 1002 \text{ SQ FT SURFACE AREA}$$

LENGTH OF TIME EXPOSED TO AIR AFTER TANK FAILURE

25 gpm pump in sump in containment area

$$\left(\frac{14{,}000 \text{ gallons}}{25 \text{ gpm}}\right) = 560 \text{ minutes} = 9.3 \text{ hours} \Rightarrow \text{say 10 hours}$$

DISTANCE TO RECEPTORS - USE SAME DISTANCE AS STORAGE TANKS

APPENDIX B
INTERPRETATION OF ASSESS OUTPUT AND ASSESS REPORTS

APPENDIX B
INTERPRETATION OF ASSESS OUTPUT AND ASSESS REPORTS

ASSESS provides the cancer risk and hazard index calculations for all exposure pathways evaluated at a site. All information needed to verify calculations is provided. Data are organized and reported in categories listed below:

- Chemical List
- Receptor/Pathway Summary
- Pathway Summary
- Intake Factors
- Media Concentrations

Numbers in ASSESS are written in scientific notation. The number following the E is a power of 10, so 1.61E-02 is 0.0161.

1.0 CHEMICAL LIST

This sheet lists the EPA toxicity values for each chemical. When available, values are listed for both oral and inhalation exposure routes for either subchronic or chronic exposure durations (reference doses or RfDs) or for carcinogenic risk (slope factors or SF). These values are obtained using the hierarchy recommended by EPA's Risk Assessment Guidance for Superfund sites (EPA 1989b, 1991a). The first and second sources in this hierarchy are (1) IRIS (Integrated Risk Information System), an on-line computer database of chemical risk information (EPA 1992d), and (2) HEAST (Health Effects Assessment Summary Tables) (EPA 1992e), EPA tables which are periodically up-dated. Toxicity values (RfDs and SFs) do not yet exist for many chemicals. For risk assessment purposes, when this is true, a chemical may be discussed qualitatively in the text of the report but will not be reported quantitatively in ASSESS.

RfDs may be derived for either subchronic or chronic exposures and are used to calculate subchronic or chronic hazard quotients, respectively. A subchronic RfD may not exist for a particular chemical even though a chronic RfD exists.

2.0 RECEPTOR/PATHWAY SUMMARY

The Receptor Summary sheet lists each receptor and the potential health risks associated with chemical exposure from all pathways quantitated. The potential adverse health risks are divided into two exposure scenarios: average exposure and reasonable maximum exposure (RME). For each exposure scenario, potential health impacts are segregated into two categories: cancer risk and a hazard index for both subchronic and chronic scenario. The values shown on this sheet show the total carcinogenic risks and noncarcinogenic hazards associated with average and RME scenario for each receptor listed.

RfDs are used with a level of exposure during a specific time period and expressed as a ratio or hazard quotient. According to the EPA (EPA 1989b, 1991a), generally, the greater the value of the hazard quotient (or hazard index), the greater the level of concern for health impacts. Another area of EPA guidance states that because H.I. is not a probability function, an increased value does not indicate a degree of risk. The hazard index is the sum of a number of hazard quotients for a particular pathway. A hazard index of one means the estimated chemical intake equals the reference dose. When this occurs, a more detailed evaluation of the site is indicated. When the H.I. is less than one, further evaluation is generally not warranted, and the exposure is expected to be safe if the H.I. is equal to or less than one.

A chemical's slope factor is multiplied by the lifetime daily average intake of that chemical in a certain medium to estimate the incremental probability (or risk) of an individual developing cancer during a lifetime. When a receptor potentially is exposed to a variety of chemicals (in a given medium) possessing slope factors, all of the products are summed to yield the total cancer risk for those chemicals for that specific exposure pathway. This risk number is acceptable if it is less than 1E-4 or 10^{-4} or 1 in 10,000.

Receptor Detail Report

This report is included in the Receptor/Pathway Summary Sheet because it details the carcinogenic risk and the noncarcinogenic hazard for each of the receptor's exposure pathway(s) for both the average and RME exposure scenarios. Total carcinogenic risks and noncarcinogenic hazards for each receptor are also given. Each receptor may have a number of exposure pathways. An exposure pathway is a route that a chemical may take to reach a population or receptor. Each pathway is categorized as one of the following:

- Inhalation
- Dermal contact with solids
- Dermal contact with water
- Ingestion of solids
- Ingestion of water

3.0 PATHWAY SUMMARY

This sheet also provides the same basic information as the Receptor Detail Report described above. The difference between the two is that the Receptor Detail Report includes all receptors while the Pathway Summary Report is receptor-specific, providing information on only one receptor per page.

Pathway Detail Report

These sheets comprise the majority of the ASSESS report. For both average and RME exposure scenarios, carcinogenic risk and noncarcinogenic hazards are listed for each receptor exposure route and chemical. Total carcinogenic risks and noncarcinogenic hazards are also provided. This report is especially helpful in determining which chemical(s) is (are) driving the overall risks or hazards for a particular exposure route. Other values provided in this report include the concentration of each chemical in the medium, the intake factor for that exposure route, and the daily intake of the chemical being evaluated. This daily intake of an individual chemical is either multiplied by its slope factor or divided by its RfD for a given exposure route (oral or inhalation). Until

other information is available from EPA, oral RfDs and SFs are used for assessing the potential health impacts of dermal exposure.

4.0 INTAKE FACTORS

These sheets represent, in equation format, the method of calculating the intake factors that appear in the pathway detail report explained above. Each of the parameters that goes into an intake factor equation is defined for both the average and RME scenarios. These parameters are more fully described in the Exposure Assessment section of this report.

5.0 MEDIA CONCENTRATIONS

These sheets describe the chemical concentrations for the average and RME scenarios for a given medium. In some cases, a higher concentration may be used to evaluate short-term risks. For example, particulate concentrations in air may be higher during a short construction project than the long-term (25 year) average might be. In most cases, the same concentrations will be used for subchronic and chronic scenario. These figures are described in the Data Summary and Selection of Chemicals of Concern section of this report.

Receptor/Pathway Summary: RMA Storage Tank RA

Receptor / Pathway	Average Exposure			Reasonable Maximum Exposure		
	Cancer Risk	Subchronic H.I.	Chronic H.I.	Cancer Risk	Subchronic H.I.	Chronic H.I.
Office Worker						
-- Inhalation of Air on-site	1.42E-08	1.10E-02	0.00E+00	3.30E-08	2.45E-02	0.00E+00
	1.42E-08	1.10E-02	0.00E+00	3.30E-08	2.45E-02	0.00E+00
Adult						
-- Inhalation of Air at Fenceline	1.67E-07	5.78E-02	0.00E+00	9.35E-07	2.91E-01	0.00E+00
	1.67E-07	5.78E-02	0.00E+00	9.35E-07	2.91E-01	0.00E+00
Child						
-- Inhalation of Air at Fenceline	4.69E-07	1.62E-01	0.00E+00	2.62E-06	8.15E-01	0.00E+00
	4.69E-07	1.62E-01	0.00E+00	2.62E-06	8.15E-01	0.00E+00

Chemical List: RMA Storage Tank RA

Chemical	Inhalation			Non-Inhalation		
	Slope Factor (mg/kg/day)-1	Subchronic RFD mg/kg/day	Chronic RFD mg/kg/day	Slope Factor (mg/kg/day)-1	Subchronic RFD mg/kg/day	Chronic RFD mg/kg/day
1,1 Dichloroethylene	1.75E-01					
1,2 Dichloropropane		3.40E-03				
Aldrin	1.70E+01					
Chlorobenzene		5.70E-02				
Chloroform	8.10E-02					
Ethyl benzene		2.93E+00				
Methylene Chloride (dichloromethane	1.64E-03	8.60E-01				
Tetrachloroethene	1.82E-03					
Toluene		1.10E-01				
Trichloroethene	6.20E-03					

Office Worker
Inhalation of Air on-site
Average Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethylene	6.20E-04	2.60E-05	1.61E-08	1.75E-01	2.82E-09	
Aldrin	2.21E-05	2.60E-05	5.74E-10	1.70E+01	9.76E-09	
Chloroform	5.88E-04	2.60E-05	1.53E-08	8.10E-02	1.24E-09	
Methylene Chloride (dichlorome	9.61E-03	2.60E-05	2.50E-07	1.64E-03	4.10E-10	
Tetrachloroethene	4.93E-05	2.60E-05	1.28E-09	1.82E-03	2.33E-12	
Trichloroethene	6.75E-05	2.60E-05	1.75E-09	6.20E-03	1.09E-11	
						1.42E-08

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	3.55E-04	9.49E-02	3.37E-05	3.40E-03	9.90E-03	
Chlorobenzene	8.14E-06	9.49E-02	7.72E-07	5.70E-02	1.35E-05	
Ethyl benzene	1.38E-05	9.49E-02	1.31E-06	2.93E+00	4.47E-07	
Methylene Chloride (dichlorome	9.61E-03	9.49E-02	9.12E-04	8.60E-01	1.06E-03	
Toluene	8.05E-06	9.49E-02	7.64E-07	1.10E-01	6.94E-06	
						1.10E-02

Office Worker
Inhalation of Air on-site
Reasonable Maximum Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethylene	8.91E-04	2.60E-05	2.32E-08	1.75E-01	4.05E-09	
Aldrin	5.76E-05	2.60E-05	1.50E-09	1.70E+01	2.54E-08	
Chloroform	7.99E-04	2.60E-05	2.08E-08	8.10E-02	1.68E-09	
Methylene Chloride (dichlorome	4.23E-02	2.60E-05	1.10E-06	1.64E-03	1.80E-09	
Tetrachloroethene	5.48E-05	2.60E-05	1.42E-09	1.82E-03	2.59E-12	
Trichloroethene	1.08E-04	2.60E-05	2.81E-09	6.20E-03	1.74E-11	
						3.30E-08

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	7.09E-04	9.49E-02	6.73E-05	3.40E-03	1.98E-02	
Chlorobenzene	2.30E-05	9.49E-02	2.18E-06	5.70E-02	3.83E-05	
Ethyl benzene	2.75E-05	9.49E-02	2.61E-06	2.93E+00	8.90E-07	
Methylene Chloride (dichlorome	4.23E-02	9.49E-02	4.01E-03	8.60E-01	4.67E-03	
Toluene	1.45E-05	9.49E-02	1.38E-06	1.10E-01	1.25E-05	
						2.45E-02

Adult
Inhalation of Air at Fenceline
Average Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethylene	1.48E-03	6.68E-05	9.89E-08	1.75E-01	1.73E-08	
Aldrin	1.23E-04	6.68E-05	8.22E-09	1.70E+01	1.40E-07	
Chloroform	1.41E-03	6.68E-05	9.42E-08	8.10E-02	7.63E-09	
Methylene Chloride (dichlorome	2.45E-02	6.68E-05	1.64E-06	1.64E-03	2.69E-09	
Tetrachloroethene	1.42E-04	6.68E-05	9.49E-09	1.82E-03	1.73E-11	
Trichloroethene	1.68E-04	6.68E-05	1.12E-08	6.20E-03	6.96E-11	
						1.67E-07

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	9.36E-04	1.90E-01	1.78E-04	3.40E-03	5.22E-02	
Chlorobenzene	2.63E-05	1.90E-01	4.99E-06	5.70E-02	8.75E-05	
Ethyl benzene	4.71E-05	1.90E-01	8.94E-06	2.93E+00	3.05E-06	
Methylene Chloride (dichlorome	2.45E-02	1.90E-01	4.65E-03	8.60E-01	5.40E-03	
Toluene	2.33E-05	1.90E-01	4.42E-06	1.10E-01	4.02E-05	
						5.78E-02

Adult
Inhalation of Air at Fenceline
Reasonable Maximum Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethylene	2.12E-03	1.51E-04	3.20E-07	1.75E-01	5.60E-08	
Aldrin	3.23E-04	1.51E-04	4.88E-08	1.70E+01	8.29E-07	
Chloroform	1.91E-03	1.51E-04	2.88E-07	8.10E-02	2.34E-08	
Methylene Chloride (dichlorome	1.07E-01	1.51E-04	1.62E-05	1.64E-03	2.65E-08	
Tetrachloroethene	1.58E-04	1.51E-04	2.39E-08	1.82E-03	4.34E-11	
Trichloroethene	2.70E-04	1.51E-04	4.08E-08	6.20E-03	2.53E-10	
						9.35E-07

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	1.88E-03	4.29E-01	8.06E-04	3.40E-03	2.37E-01	
Chlorobenzene	7.44E-05	4.29E-01	3.19E-05	5.70E-02	5.59E-04	
Ethyl benzene	9.43E-05	4.29E-01	4.04E-05	2.93E+00	1.38E-05	
Methylene Chloride (dichlorome	1.07E-01	4.29E-01	4.59E-02	8.60E-01	5.33E-02	
Toluene	4.19E-05	4.29E-01	1.80E-05	1.10E-01	1.63E-04	
						2.91E-01

Child
Inhalation of Air at Fenceline
Average Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethylene	1.48E-03	1.87E-04	2.77E-07	1.75E-01	4.85E-08	
Aldrin	1.23E-04	1.87E-04	2.30E-08	1.70E+01	3.91E-07	
Chloroform	1.41E-03	1.87E-04	2.64E-07	8.10E-02	2.14E-08	
Methylene Chloride (dichlorome	2.45E-02	1.87E-04	4.58E-06	1.64E-03	7.52E-09	
Tetrachloroethene	1.42E-04	1.87E-04	2.66E-08	1.82E-03	4.84E-11	
Trichloroethene	1.68E-04	1.87E-04	3.14E-08	6.20E-03	1.95E-10	
						4.69E-07

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	9.36E-04	5.31E-01	4.97E-04	3.40E-03	1.46E-01	
Chlorobenzene	2.63E-05	5.31E-01	1.40E-05	5.70E-02	2.45E-04	
Ethyl benzene	4.71E-05	5.31E-01	2.50E-05	2.93E+00	8.54E-06	
Methylene Chloride (dichlorome	2.45E-02	5.31E-01	1.30E-02	8.60E-01	1.51E-02	
Toluene	2.33E-05	5.31E-01	1.24E-05	1.10E-01	1.13E-04	
						1.62E-01

Child
Inhalation of Air at Fenceline
Reasonable Maximum Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethylene	2.12E-03	4.23E-04	8.96E-07	1.75E-01	1.57E-07	
Aldrin	3.23E-04	4.23E-04	1.37E-07	1.70E+01	2.32E-06	
Chloroform	1.91E-03	4.23E-04	8.07E-07	8.10E-02	6.54E-08	
Methylene Chloride (dichlorome	1.07E-01	4.23E-04	4.52E-05	1.64E-03	7.42E-08	
Tetrachloroethene	1.58E-04	4.23E-04	6.68E-08	1.82E-03	1.22E-10	
Trichloroethene	2.70E-04	4.23E-04	1.14E-07	6.20E-03	7.08E-10	
						2.62E-06

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	1.88E-03	1.20E+00	2.26E-03	3.40E-03	6.64E-01	
Chlorobenzene	7.44E-05	1.20E+00	8.93E-05	5.70E-02	1.57E-03	
Ethyl benzene	9.43E-05	1.20E+00	1.13E-04	2.93E+00	3.86E-05	
Methylene Chloride (dichlorome	1.07E-01	1.20E+00	1.28E-01	8.60E-01	1.49E-01	
Toluene	4.19E-05	1.20E+00	5.03E-05	1.10E-01	4.57E-04	
						8.15E-01

Intake Factor: RMA Storage Tank RA

Description: Office Worker On-siite
Abbreviation: OOVI
Pathway Type: Inhalation

$$\text{Intake Factor} = \frac{(\text{IR} \times \text{ET} \times \text{EF} \times \text{ED} \times \text{ME} \times \text{DF} \times \text{CC} \times \text{FI} \times \text{SS})}{(\text{BW} \times \text{AT})}$$

Parameters:

Abbreviation	Description	Carcinogenic Ave	Carcinogenic RME	Non-Carcinogenic Ave	Non-Carcinogenic RME
IR	Inhalation Rate(m3/hr)	.83	.83	.83	.83
ET	Exposure Time(hrs/dy)	8	8	8	8
EF	Exposure Freq.(dys/yr)	7	7	7	7
ED	Exposure Duration(yrs)	1	1	1	1
ME	Matrix Effect	1	1	1	1
DF	Deposition Factor	1	1	1	1
CC	Ciliary Clearance	1	1	1	1
FI	Fraction Contaminated	1	1	1	1
SS	Site-Specific Factor	1	1	1	1
BW	Body Weight(kg)	70	70	70	70
AT	Averaging Time(dys)	25550	25550	7	7

Intake Factor Calculations:

Carcinogenic: Average Exposure

$$2.60E\text{-}05 = \frac{(8.30E\text{-}01 \times 8.00E{+}00 \times 7.00E{+}00 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E{+}01 \times 2.56E{+}04)}$$

Carcinogenic: Reasonable Maximum Exposure

$$2.60E\text{-}05 = \frac{(8.30E\text{-}01 \times 8.00E{+}00 \times 7.00E{+}00 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E{+}01 \times 2.56E{+}04)}$$

Non-Carcinogenic: Average Exposure

$$9.49E\text{-}02 = \frac{(8.30E\text{-}01 \times 8.00E{+}00 \times 7.00E{+}00 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E{+}01 \times 7.00E{+}00)}$$

Non-Carcinogenic: Reasonable Maximum Exposure

$$9.49E\text{-}02 = \frac{(8.30E\text{-}01 \times 8.00E{+}00 \times 7.00E{+}00 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E{+}01 \times 7.00E{+}00)}$$

Intake Factor: RMA Storage Tank RA

Description: Adult at Fenceline
Abbreviation: AFVI
Pathway Type: Inhalation

$$\text{Intake Factor} = \frac{(\text{IR} \times \text{ET} \times \text{EF} \times \text{ED} \times \text{ME} \times \text{DF} \times \text{CC} \times \text{FI} \times \text{SS})}{(\text{BW} \times \text{AT})}$$

Parameters:

Abbreviation	Description	Carcinogenic Ave	Carcinogenic RME	Non-Carcinogenic Ave	Non-Carcinogenic RME
IR	Inhalation Rate(m3/hr)	.83	1.25	.83	1.25
ET	Exposure Time(hrs/dy)	16	24	16	24
EF	Exposure Freq.(dys/yr)	9	9	9	9
ED	Exposure Duration(yrs)	1	1	1	1
ME	Matrix Effect	1	1	1	1
DF	Deposition Factor	1	1	1	1
CC	Ciliary Clearance	1	1	1	1
FI	Fraction Contaminated	1	1	1	1
SS	Site-Specific Factor	1	1	1	1
BW	Body Weight(kg)	70	70	70	70
AT	Averaging Time(dys)	25550	25550	9	9

Intake Factor Calculations:

Carcinogenic: Average Exposure

$$6.68E\text{-}05 = \frac{(8.30E\text{-}01 \times 1.60E{+}01 \times 9.00E{+}00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E{+}01 \times 2.56E{+}04)}$$

Carcinogenic: Reasonable Maximum Exposure

$$1.51E\text{-}04 = \frac{(1.25E{+}00 \times 2.40E{+}01 \times 9.00E{+}00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E{+}01 \times 2.56E{+}04)}$$

Non-Carcinogenic: Average Exposure

$$1.90E\text{-}01 = \frac{(8.30E\text{-}01 \times 1.60E{+}01 \times 9.00E{+}00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E{+}01 \times 9.00E{+}00)}$$

Non-Carcinogenic: Reasonable Maximum Exposure

$$4.29E\text{-}01 = \frac{(1.25E{+}00 \times 2.40E{+}01 \times 9.00E{+}00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E{+}01 \times 9.00E{+}00)}$$

Intake Factor: RMA Storage Tank RA

Description: Child at Fenceline
Abbreviation: CFVI
Pathway Type: Inhalation

$$\text{Intake Factor} = \frac{(\text{IR} \times \text{ET} \times \text{EF} \times \text{ED} \times \text{ME} \times \text{DF} \times \text{CC} \times \text{FI} \times \text{SS})}{(\text{BW} \times \text{AT})}$$

Parameters:

Abbreviation	Description	Carcinogenic Ave	Carcinogenic RME	Non-Carcinogenic Ave	Non-Carcinogenic RME
IR	Inhalation Rate(m3/hr)	.83	1.25	.83	1.25
ET	Exposure Time(hrs/dy)	16	24	16	24
EF	Exposure Freq.(dys/yr)	9	9	9	9
ED	Exposure Duration(yrs)	1	1	1	1
ME	Matrix Effect	1	1	1	1
DF	Deposition Factor	1	1	1	1
CC	Ciliary Clearance	1	1	1	1
FI	Fraction Contaminated	1	1	1	1
SS	Site-Specific Factor	1	1	1	1
BW	Body Weight(kg)	25	25	25	25
AT	Averaging Time(dys)	25550	25550	9	9

Intake Factor Calculations:

Carcinogenic: Average Exposure

$$1.87\text{E-}04 = \frac{(8.30\text{E-}01 \times 1.60\text{E+}01 \times 9.00\text{E+}00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(2.50\text{E+}01 \times 2.56\text{E+}04)}$$

Carcinogenic: Reasonable Maximum Exposure

$$4.23\text{E-}04 = \frac{(1.25\text{E+}00 \times 2.40\text{E+}01 \times 9.00\text{E+}00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(2.50\text{E+}01 \times 2.56\text{E+}04)}$$

Non-Carcinogenic: Average Exposure

$$5.31\text{E-}01 = \frac{(8.30\text{E-}01 \times 1.60\text{E+}01 \times 9.00\text{E+}00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(2.50\text{E+}01 \times 9.00\text{E+}00)}$$

Non-Carcinogenic: Reasonable Maximum Exposure

$$1.20\text{E+}00 = \frac{(1.25\text{E+}00 \times 2.40\text{E+}01 \times 9.00\text{E+}00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(2.50\text{E+}01 \times 9.00\text{E+}00)}$$

Media Chemical Concentrations: RMA Storage Tank RA

Medium: Air on-site
Abbreviation: Air1
Medium Type: Particulate / VOCs

Chemical	Subchronic		Chronic/Carcinogenic	
	Average (mg/m3)	R.M.E. (mg/m3)	Average (mg/m3)	R.M.E. (mg/m3)
1,1 Dichloroethylene	6.20E-04	8.91E-04	6.20E-04	8.91E-04
1,2 Dichloropropane	3.55E-04	7.09E-04	3.55E-04	7.09E-04
Aldrin	2.21E-05	5.76E-05	2.21E-05	5.76E-05
Chlorobenzene	8.14E-06	2.30E-05	8.14E-06	2.30E-05
Chloroform	5.88E-04	7.99E-04	5.88E-04	7.99E-04
Ethyl benzene	1.38E-05	2.75E-05	1.38E-05	2.75E-05
Methylene Chloride (dichloromethane	9.61E-03	4.23E-02	9.61E-03	4.23E-02
Tetrachloroethene	4.93E-05	5.48E-05	4.93E-05	5.48E-05
Toluene	8.05E-06	1.45E-05	8.05E-06	1.45E-05
Trichloroethene	6.75E-05	1.08E-04	6.75E-05	1.08E-04

Media Chemical Concentrations: RMA Storage Tank RA

Medium: Air at Fenceline
Abbreviation: Air2
Medium Type: Particulate / VOCs

Chemical	Subchronic		Chronic/Carcinogenic	
	Average (mg/m3)	R.M.E. (mg/m3)	Average (mg/m3)	R.M.E. (mg/m3)
1,1 Dichloroethylene	1.48E-03	2.12E-03	1.48E-03	2.12E-03
1,2 Dichloropropane	9.36E-04	1.88E-03	9.36E-04	1.88E-03
Aldrin	1.23E-04	3.23E-04	1.23E-04	3.23E-04
Chlorobenzene	2.63E-05	7.44E-05	2.63E-05	7.44E-05
Chloroform	1.41E-03	1.91E-03	1.41E-03	1.91E-03
Ethyl benzene	4.71E-05	9.43E-05	4.71E-05	9.43E-05
Methylene Chloride (dichloromethane	2.45E-02	1.07E-01	2.45E-02	1.07E-01
Tetrachloroethene	1.42E-04	1.58E-04	1.42E-04	1.58E-04
Toluene	2.33E-05	4.19E-05	2.33E-05	4.19E-05
Trichloroethene	1.68E-04	2.70E-04	1.68E-04	2.70E-04

Receptor/Pathway Summary: RMA Day Storage Tank RA

March 29, 1993
15:13

Receptor / Pathway	Average Exposure			Reasonable Maximum Exposure		
	Cancer Risk	Subchronic H.I.	Chronic H.I.	Cancer Risk	Subchronic H.I.	Chronic H.I.
Office Worker						
-- Inhalation of Air On-site	3.26E-10	3.69E-04	0.00E+00	8.37E-10	7.92E-04	0.00E+00
	3.26E-10	3.69E-04	0.00E+00	8.37E-10	7.92E-04	0.00E+00
Adult						
-- Inhalation of Air at Fenceline	2.24E-09	2.06E-03	0.00E+00	8.71E-09	6.11E-03	0.00E+00
	2.24E-09	2.06E-03	0.00E+00	8.71E-09	6.11E-03	0.00E+00
Child						
-- Inhalation of Air at Fenceline	6.26E-09	5.43E-03	0.00E+00	2.44E-08	1.71E-02	0.00E+00
	6.26E-09	5.43E-03	0.00E+00	2.44E-08	1.71E-02	0.00E+00

Office Worker
Inhalation of Air On-site
Average Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethene	1.28E-05	3.71E-06	4.75E-11	1.75E-01	8.32E-12	
Aldrin	4.95E-06	3.71E-06	1.84E-11	1.70E+01	3.12E-10	
Chloroform	1.26E-05	3.71E-06	4.68E-11	8.10E-02	3.79E-12	
Methylene Chloride	1.98E-04	3.71E-06	7.35E-10	1.64E-03	1.21E-12	
Tetrachloroethene	2.40E-06	3.71E-06	8.91E-12	1.82E-03	1.62E-14	
Trichloroethene	1.97E-06	3.71E-06	7.31E-12	6.20E-03	4.53E-14	
						3.26E-10

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	1.24E-05	9.49E-02	1.18E-06	3.40E-03	3.46E-04	
Chlorobenzene	6.13E-07	9.49E-02	5.81E-08	5.70E-02	1.02E-06	
Ethyl benzene	1.20E-06	9.49E-02	1.14E-07	2.93E+00	3.88E-08	
Methylene Chloride	1.98E-04	9.49E-02	1.88E-05	8.60E-01	2.18E-05	
Toluene	4.16E-07	9.49E-02	3.95E-08	1.10E-01	3.59E-07	
						3.69E-04

Office Worker
Inhalation of Air On-site
Reasonable Maximum Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethene	1.84E-05	3.71E-06	6.83E-11	1.75E-01	1.20E-11	
Aldrin	1.29E-05	3.71E-06	4.79E-11	1.70E+01	8.14E-10	
Chloroform	1.71E-05	3.71E-06	6.35E-11	8.10E-02	5.14E-12	
Methylene Chloride	8.71E-04	3.71E-06	3.23E-09	1.64E-03	5.30E-12	
Tetrachloroethene	2.66E-06	3.71E-06	9.88E-12	1.82E-03	1.80E-14	
Trichloroethene	3.08E-06	3.71E-06	1.14E-11	6.20E-03	7.09E-14	
						8.37E-10

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	2.48E-05	9.49E-02	2.35E-06	3.40E-03	6.92E-04	
Chlorobenzene	1.74E-06	9.49E-02	1.65E-07	5.70E-02	2.90E-06	
Ethyl benzene	2.39E-06	9.49E-02	2.27E-07	2.93E+00	7.74E-08	
Methylene Chloride	8.71E-04	9.49E-02	8.26E-05	8.60E-01	9.61E-05	
Toluene	7.48E-07	9.49E-02	7.10E-08	1.10E-01	6.45E-07	
						7.92E-04

Adult
Inhalation of Air at Fenceline
Average Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethene	3.96E-05	4.64E-06	1.84E-10	1.75E-01	3.22E-11	
Aldrin	2.77E-05	4.64E-06	1.29E-10	1.70E+01	2.19E-09	
Chloroform	4.00E-05	4.64E-06	1.86E-10	8.10E-02	1.50E-11	
Methylene Chloride	5.58E-04	4.64E-06	2.59E-09	1.64E-03	4.25E-12	
Tetrachloroethene	1.13E-05	4.64E-06	5.24E-11	1.82E-03	9.54E-14	
Trichloroethene	7.28E-06	4.64E-06	3.38E-11	6.20E-03	2.09E-13	
						2.24E-09

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	5.32E-05	1.26E-01	6.71E-06	3.40E-03	1.97E-03	
Chlorobenzene	3.08E-06	1.26E-01	3.89E-07	5.70E-02	6.82E-06	
Ethyl benzene	6.13E-06	1.26E-01	7.73E-07	2.93E+00	2.64E-07	
Methylene Chloride	5.58E-04	1.26E-01	7.04E-05	8.60E-01	8.18E-05	
Toluene	1.94E-06	1.26E-01	2.45E-07	1.10E-01	2.22E-06	
						2.06E-03

Adult
Inhalation of Air at Fenceline
Reasonable Maximum Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethene	5.68E-05	6.99E-06	3.97E-10	1.75E-01	6.95E-11	
Aldrin	7.22E-05	6.99E-06	5.05E-10	1.70E+01	8.58E-09	
Chloroform	5.43E-05	6.99E-06	3.80E-10	8.10E-02	3.07E-11	
Methylene Chloride	2.46E-03	6.99E-06	1.72E-08	1.64E-03	2.82E-11	
Tetrachloroethene	1.25E-05	6.99E-06	8.74E-11	1.82E-03	1.59E-13	
Trichloroethene	1.17E-05	6.99E-06	8.18E-11	6.20E-03	5.07E-13	
						8.71E-09

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	1.06E-04	1.79E-01	1.89E-05	3.40E-03	5.57E-03	
Chlorobenzene	8.73E-06	1.79E-01	1.56E-06	5.70E-02	2.73E-05	
Ethyl benzene	1.23E-05	1.79E-01	2.20E-06	2.93E+00	7.50E-07	
Methylene Chloride	2.46E-03	1.79E-01	4.39E-04	8.60E-01	5.11E-04	
Toluene	3.50E-06	1.79E-01	6.25E-07	1.10E-01	5.68E-06	
						6.11E-03

Child
Inhalation of Air at Fenceline
Average Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethene	3.96E-05	1.30E-05	5.15E-10	1.75E-01	9.00E-11	
Aldrin	2.77E-05	1.30E-05	3.60E-10	1.70E+01	6.12E-09	
Chloroform	4.00E-05	1.30E-05	5.20E-10	8.10E-02	4.21E-11	
Methylene Chloride	5.58E-04	1.30E-05	7.25E-09	1.64E-03	1.19E-11	
Tetrachloroethene	1.13E-05	1.30E-05	1.47E-10	1.82E-03	2.67E-13	
Trichloroethene	7.28E-06	1.30E-05	9.46E-11	6.20E-03	5.87E-13	
						6.26E-09

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	5.32E-05	3.32E-01	1.77E-05	3.40E-03	5.19E-03	
Chlorobenzene	3.08E-06	3.32E-01	1.02E-06	5.70E-02	1.79E-05	
Ethyl benzene	6.13E-06	3.32E-01	2.04E-06	2.93E+00	6.95E-07	
Methylene Chloride	5.58E-04	3.32E-01	1.85E-04	8.60E-01	2.15E-04	
Toluene	1.94E-06	3.32E-01	6.44E-07	1.10E-01	5.86E-06	
						5.43E-03

Child
Inhalation of Air at Fenceline
Reasonable Maximum Exposure

Carcinogenic Risk

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Slope Factor (mg/kg/day)-1	Carcinogenic Risk	Total Risk
1,1 Dichloroethene	5.68E-05	1.96E-05	1.11E-09	1.75E-01	1.95E-10	
Aldrin	7.22E-05	1.96E-05	1.41E-09	1.70E+01	2.40E-08	
Chloroform	5.43E-05	1.96E-05	1.06E-09	8.10E-02	8.61E-11	
Methylene Chloride	2.46E-03	1.96E-05	4.81E-08	1.64E-03	7.90E-11	
Tetrachloroethene	1.25E-05	1.96E-05	2.45E-10	1.82E-03	4.45E-13	
Trichloroethene	1.17E-05	1.96E-05	2.29E-10	6.20E-03	1.42E-12	
						2.44E-08

Hazard Index -- Subchronic

Chemical	Chemical Concentration (mg/m3)	Inhalation Intake Factor (m3/kg/day)	Daily Intake (mg/kg/day)	Subchronic RFD (mg/kg/day)	Hazard Quotient	Hazard Index
1,2 Dichloropropane	1.06E-04	5.00E-01	5.30E-05	3.40E-03	1.56E-02	
Chlorobenzene	8.73E-06	5.00E-01	4.37E-06	5.70E-02	7.66E-05	
Ethyl benzene	1.23E-05	5.00E-01	6.15E-06	2.93E+00	2.10E-06	
Methylene Chloride	2.46E-03	5.00E-01	1.23E-03	8.60E-01	1.43E-03	
Toluene	3.50E-06	5.00E-01	1.75E-06	1.10E-01	1.59E-05	
						1.71E-02

Description: Office Worker-Inhalation of VOCs on-site
Abbreviation: OOVI
Pathway Type: Inhalation

$$\text{Intake Factor} = \frac{(\text{IR} \times \text{ET} \times \text{EF} \times \text{ED} \times \text{ME} \times \text{DF} \times \text{CC} \times \text{FI} \times \text{SS})}{(\text{BW} \times \text{AT})}$$

Parameters:

Abbreviation	Description	Carcinogenic Ave	Carcinogenic RME	Non-Carcinogenic Ave	Non-Carcinogenic RME
IR	Inhalation Rate(m3/hr)	.83	.83	.83	.83
ET	Exposure Time(hrs/dy)	8	8	8	8
EF	Exposure Freq.(dys/yr)	1	1	1	1
ED	Exposure Duration(yrs)	1	1	1	1
ME	Matrix Effect	1	1	1	1
DF	Deposition Factor	1	1	1	1
CC	Ciliary Clearance	1	1	1	1
FI	Fraction Contaminated	1	1	1	1
SS	Site-Specific Factor	1	1	1	1
BW	Body Weight(kg)	70	70	70	70
AT	Averaging Time(dys)	25550	25550	1	1

Intake Factor Calculations:

Carcinogenic: Average Exposure

$$3.71E\text{-}06 = \frac{(8.30E\text{-}01 \times 8.00E+00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E+01 \times 2.56E+04)}$$

Carcinogenic: Reasonable Maximum Exposure

$$3.71E\text{-}06 = \frac{(8.30E\text{-}01 \times 8.00E+00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E+01 \times 2.56E+04)}$$

Non-Carcinogenic: Average Exposure

$$9.49E\text{-}02 = \frac{(8.30E\text{-}01 \times 8.00E+00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E+01 \times 1)}$$

Non-Carcinogenic: Reasonable Maximum Exposure

$$9.49E\text{-}02 = \frac{(8.30E\text{-}01 \times 8.00E+00 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00E+01 \times 1)}$$

Description: Adult Resident-VOC inhalation at fncline
Abbreviation: ARVI
Pathway Type: Inhalation

$$\text{Intake Factor} = \frac{(\text{IR} \times \text{ET} \times \text{EF} \times \text{ED} \times \text{ME} \times \text{DF} \times \text{CC} \times \text{FI} \times \text{SS})}{(\text{BW} \times \text{AT})}$$

Parameters:

Abbreviation	Description	Carcinogenic Ave	Carcinogenic RME	Non-Carcinogenic Ave	Non-Carcinogenic RME
IR	Inhalation Rate(m3/hr)	.83	1.25	.883	1.25
ET	Exposure Time(hrs/dy)	10	10	10	10
EF	Exposure Freq.(dys/yr)	1	1	1	1
ED	Exposure Duration(yrs)	1	1	1	1
ME	Matrix Effect	1	1	1	1
DF	Deposition Factor	1	1	1	1
CC	Ciliary Clearance	1	1	1	1
FI	Fraction Contaminated	1	1	1	1
SS	Site-Specific Factor	1	1	1	1
BW	Body Weight(kg)	70	70	70	70
AT	Averaging Time(dys)	25550	25550	1	1

Intake Factor Calculations:

Carcinogenic: Average Exposure

$$4.64\text{E-}06 = \frac{(8.30\text{E-}01 \times 1.00\text{E+}01 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00\text{E+}01 \times 2.56\text{E+}04)}$$

Carcinogenic: Reasonable Maximum Exposure

$$6.99\text{E-}06 = \frac{(1.25\text{E+}00 \times 1.00\text{E+}01 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00\text{E+}01 \times 2.56\text{E+}04)}$$

Non-Carcinogenic: Average Exposure

$$1.26\text{E-}01 = \frac{(8.83\text{E-}01 \times 1.00\text{E+}01 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00\text{E+}01 \times 1)}$$

Non-Carcinogenic: Reasonable Maximum Exposure

$$1.79\text{E-}01 = \frac{(1.25\text{E+}00 \times 1.00\text{E+}01 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(7.00\text{E+}01 \times 1)}$$

Intake Factor: RMA Day Storage Tank RA

March 29, 1993
15:13

Description: Child Resident-VOC Inhalation at fncline
Abbreviation: CRVI
Pathway Type: Inhalation

$$\text{Intake Factor} = \frac{(\text{IR} \times \text{ET} \times \text{EF} \times \text{ED} \times \text{ME} \times \text{DF} \times \text{CC} \times \text{FI} \times \text{SS})}{(\text{BW} \times \text{AT})}$$

Parameters:

Abbreviation	Description	Carcinogenic Ave	Carcinogenic RME	Non-Carcinogenic Ave	Non-Carcinogenic RME
IR	Inhalation Rate(m3/hr)	.83	1.25	.83	1.25
ET	Exposure Time(hrs/dy)	10	10	10	10
EF	Exposure Freq.(dys/yr)	1	1	1	1
ED	Exposure Duration(yrs)	1	1	1	1
ME	Matrix Effect	1	1	1	1
DF	Deposition Factor	1	1	1	1
CC	Ciliary Clearance	1	1	1	1
FI	Fraction Contaminated	1	1	1	1
SS	Site-Specific Factor	1	1	1	1
BW	Body Weight(kg)	25	25	25	25
AT	Averaging Time(dys)	25550	25550	1	1

Intake Factor Calculations:

Carcinogenic: Average Exposure

$$1.30\text{E-}05 = \frac{(8.30\text{E-}01 \times 1.00\text{E+}01 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(2.50\text{E+}01 \times 2.56\text{E+}04)}$$

Carcinogenic: Reasonable Maximum Exposure

$$1.96\text{E-}05 = \frac{(1.25\text{E+}00 \times 1.00\text{E+}01 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(2.50\text{E+}01 \times 2.56\text{E+}04)}$$

Non-Carcinogenic: Average Exposure

$$3.32\text{E-}01 = \frac{(8.30\text{E-}01 \times 1.00\text{E+}01 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(2.50\text{E+}01 \times 1)}$$

Non-Carcinogenic: Reasonable Maximum Exposure

$$5.00\text{E-}01 = \frac{(1.25\text{E+}00 \times 1.00\text{E+}01 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1 \times 1)}{(2.50\text{E+}01 \times 1)}$$

Medium: Air On-site
Abbreviation: Air1
Medium Type: Particulate / VOCs

Chemical	Subchronic		Chronic/Carcinogenic	
	Average (mg/m3)	R.M.E. (mg/m3)	Average (mg/m3)	R.M.E. (mg/m3)
1,1 Dichloroethene	1.28E-05	1.84E-05	1.28E-05	1.84E-05
1,2 Dichloropropane	1.24E-05	2.48E-05	1.24E-05	2.48E-05
Aldrin	4.95E-06	1.29E-05	4.95E-06	1.29E-05
Chlorobenzene	6.13E-07	1.74E-06	6.13E-07	1.74E-06
Chloroform	1.26E-05	1.71E-05	1.26E-05	1.71E-05
Ethyl benzene	1.20E-06	2.39E-06	1.20E-06	2.39E-06
Methylene Chloride	1.98E-04	8.71E-04	1.98E-04	8.71E-04
Tetrachloroethene	2.40E-06	2.66E-06	2.40E-06	2.66E-06
Toluene	4.16E-07	7.48E-07	4.16E-07	7.48E-07
Trichloroethene	1.97E-06	3.08E-06	1.97E-06	3.08E-06

Medium: Air at Fenceline
Abbreviation: Air2
Medium Type: Particulate / VOCs

Chemical	Subchronic		Chronic/Carcinogenic	
	Average (mg/m3)	R.M.E. (mg/m3)	Average (mg/m3)	R.M.E. (mg/m3)
1,1 Dichloroethene	3.96E-05	5.68E-05	3.96E-05	5.68E-05
1,2 Dichloropropane	5.32E-05	1.06E-04	5.32E-05	1.06E-04
Aldrin	2.77E-05	7.22E-05	2.77E-05	7.22E-05
Chlorobenzene	3.08E-06	8.73E-06	3.08E-06	8.73E-06
Chloroform	4.00E-05	5.43E-05	4.00E-05	5.43E-05
Ethyl benzene	6.13E-06	1.23E-05	6.13E-06	1.23E-05
Methylene Chloride	5.58E-04	2.46E-03	5.58E-04	2.46E-03
Tetrachloroethene	1.13E-05	1.25E-05	1.13E-05	1.25E-05
Toluene	1.94E-06	3.50E-06	1.94E-06	3.50E-06
Trichloroethene	7.28E-06	1.17E-05	7.28E-06	1.17E-05

Chemical	Inhalation			Non-Inhalation		
	Slope Factor (mg/kg/day)-1	Subchronic RFD mg/kg/day	Chronic RFD mg/kg/day	Slope Factor (mg/kg/day)-1	Subchronic RFD mg/kg/day	Chronic RFD mg/kg/day
1,1 Dichloroethene	1.75E-01					
1,2 Dichloropropane		3.40E-03				
Aldrin	1.70E+01					
Chlorobenzene		5.70E-02				
Chloroform	8.10E-02					
Ethyl benzene		2.93E+00				
Methylene Chloride	1.64E-03	8.60E-01				
Tetrachloroethene	1.82E-03					
Toluene		1.10E-01				
Trichloroethene	6.20E-03					

APPENDIX C
TOXICITY PROFILES

APPENDIX C
TOXICITY PROFILES

1,1-DICHLOROETHYLENE

1,1 Dichloroethylene (1,1-DCE), or vinylidene chloride, is a common intermediate in the manufacture of polymers. 1,1-DCE polymers are widely used as coatings on the interiors of railroad cars, fuel storage tanks, and ship tanks, and on steel pipes and structures.

The chemical is highly volatile. 1,1-DCE is expected to be short-lived in water because of its poor solubility and rapid volatilization. Its half-life in surface water is estimated at 1 to 6 days, depending on aeration rates. Its volatility and poor solubility probably prevents absorption of significant quantities through skin.

Data on acute oral toxicity in animal studies, as measured by the LD_{50} (Lethal Dose) of 50 mg/kg to 1800 mg/kg in rats and 5750 mg/kg in dogs have been reported. Acute oral doses were found to produce numerous changes in liver and plasma enzymes as well as cell damage in liver and the bronchial epithelium.

Chronic (2-year) oral exposures via drinking water at 1,1-DCE concentrations of 50, 100, or 200 mg/l produced signs of liver pathology in rats. Chronic renal inflammation was observed in rats given 5 mg/kg/day by gavage for 2 years, and liver necrosis has been produced in mice exposed to 10 mg/kg/day by gavage, 5 days/week for 2 years. No adverse effects were observed at lower doses.

Acute inhalation toxicity in rats, measured as LC_{50} (Lethal Concentration) following 4 hours of exposure, also varied with whether the animal was fed (15,000 ppm) or fasted (600 ppm). Results from other studies are variable. For example, rabbits, monkeys, rats, and guinea pigs exposed to 395 mg/m^3 (100 ppm) for 8 hours/day, 5 days/week for 6 weeks exhibited no mortality, signs of toxicity, or histopathological (tissue) changes. Several studies have demonstrated that species, strain, and sex greatly influence the acute toxic effects of 1,1-DCE.

Longer term inhalation exposures, either continuous (47 ppm, 90 days) or intermittent (50 to 100 ppm, 8 hours/day, 5 days/week for 6 months), have been shown to produce liver, kidney, and lung damage in some animals.

If microsomal activation is provided, 1,1-DCE is weakly mutagenic in some bacterial systems, but not in Chinese hamster cells or in mice and rats. The International Agency for Research on Cancer has concluded that the evidence is sufficient to classify 1,1-DCE as a mutagen.

Studies of carcinogenic effects have variable results. Several long-term studies in rats failed to produce any evidence of carcinogenicity, but increased incidence of kidney and mammary tumors have been reported in Swiss mice. However, the tumors may be the result of tissue injury and repair rather than the result of mechanisms involving DNA.

Based on the lack of evidence of carcinogenicity in humans, limited animal data, and the mutagenicity observed in bacterial systems, EPA has classified 1,1-DCE as a possible human carcinogen (Class C). EPA has published an oral slope factor of 6E-1 $(mg/kg/day)^{-1}$ and a drinking water unit risk of 1.75E-5 $(\mu g/l)^{-1}$ based on a National Toxicology Program study that showed no increase in rat adrenal tumors at doses equivalent to a human intake of 0.6 mg/kg/day.

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1,2-DICHLOROETHANE

The majority of 1,2-dichloroethane (1,2-DCA) use is for the production of vinyl chloride. It is also used as a gasoline additive, fumigant or solvent. The toxic effects of 1,2-DCA are similar in animals and humans. These effects are largely the result of central nervous system depression (confusion, dizziness, nausea, vomiting, convulsions). Effects via oral and inhalation routes are similar. At high concentrations, 1,2-DCA is irritating to the eyes, nose and throat. Chronic exposure can result in injury to the kidney, liver, and adrenal glands. The EPA weight of evidence for the carcinogenic potency of 1,2-DCA is Group B2 (probable human carcinogen) based on the induction of several tumor types in rats and mice treated by gavage and lung papillomas in mice after topical application.

At subacute levels, similar symptoms of CNS depression and gastrointestinal upset are observed. Definite liver, kidney, and adrenal injury may occur at these levels (Clayton and Clayton). The symptom of nausea and vomiting from this chemical is strikingly similar to that often seen from carbon tetrachloride exposure. (La Dou 1990). The overarching effects of 1,2-dichloroethane in animals has also been relegated to visual organ damage and CNS effects.

Inhalation of 1,2-dichloroethane in animals also has similar CNS effects. (ATSDR 1989).

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DIELDRIN (AND ALDRIN)

Dieldrin and aldrin are synthetic organic compounds first introduced in the 1950s as pesticides, but since 1985 they have no longer been produced in the United States. They are registered for use in the United States to control subterranean termites, for the dipping of nonfood plant roots and tops to control insect infestations, and for moth-proofing during manufacturing processes in a closed system. The two latter uses have been voluntarily curtailed by industry. Use of dieldrin for termite control is currently allowed until supplies available before 1985 are exhausted.

Dieldrin and Aldrin are structurally quite similar and are often used in combination with each other. In fact, aldrin is readily converted via epoxidation into dieldrin in the body (Bann et al. 1956). Thus, the effects of aldrin and dieldrin are quite similar in both experimental animals and humans (Hayes 1963) and will be discussed in tandem.

Dieldrin can be absorbed into the blood from the gastrointestinal tract, through the skin, and through the lungs. The percentage of oral dose absorption has not been accurately determined. In humans, approximately 20 to 50 percent of inhaled dieldrin is retained and about 8 percent of a dermal dose is absorbed. Absorbed aldrin and dieldrin are distributed to the liver and other tissues and are accumulated in body fat. Dieldrin in fat can be mobilized to the liver where it is metabolized. Excretion occurs primarily through feces via the bile. Urinary excretion is minor.

Studies on humans exposed to dieldrin indicate that .2 mg/ml of blood is the approximate threshold at which symptoms of intoxication occur (USEPA 1980). The oral TD_{LO} required to produce central nervous system (CNS) effects in humans is 14 mg/kg (USEPA 1980). In a somewhat contradictory study of 12 human males, each was given oral doses of pure dieldrin in concentrations ranging from 0 to 211 mg/kg/day for periods of 6 months to 24 months. An observation period of 8 months' post-treatment revealed no signs of toxicity or adverse effects with respect to body weight, EEGs, ECGs, hematologic parameters, or serum enzyme activities (USEPA 1980). It is not known whether these parameters coincide with those by which the TD_{LO} given above was calculated.

Dieldrin and Aldrin are both acutely toxic to laboratory animals by the oral, dermal, and inhalation routes. Both are mildly irritating to the eye and to the skin. The acute oral LD_{50} of aldrin and dieldrin in experimental animals (including rats, mice, dogs, monkeys, and sheep) ranges from 20 mg/kg to 70 mg/kg (Hodge et al. 1967).

Results of studies on the teratogenicity of aldrin and dieldrin have been variable. Dieldrin and Aldrin exposures are reportedly associated with effects on reproduction (i.e., decreased fertility and decreased viability of the offspring) in a variety of species, but the dietary concentrations required for these effects were as high or higher than those which produced toxic effects in adult animals. An increase in fetal death and fetal malformation were observed in Syrian golden hamsters following a single oral dose of 50 mg/kg aldrin or 30 mg/kg dieldrin (USEPA 1980). Mice treated with aldrin and dieldrin at 25 mg/kg or 15 mg/kg, respectively, in the same study showed no effect on fetal weight or survival, although terata were observed.

Dieldrin has been reported to cause immunosuppression in laboratory animals. Mice exposed to dieldrin and challenged with Leishmania tropica had decreased antibody formation and reduced mitogenic response (ATSDR 1987). Other studies have shown that dieldrin impairs antigen processing and reduces host resistance to mouse hepatitis virus (ATSDR 1987).

Four chronic feeding studies investigating the carcinogenicity of aldrin and dieldrin in rats have yielded negative results (Deichman et al. 1970; Walker et al. 1969; Deichman et al. 1967; Cleveland 1966). Other studies in rats revealed no statistically significant increases in hepatocarcinomas after exposure to aldrin (29 or 65 mg/kg) or dieldrin (29 or 65 mg/kg) in the diet (NCI 1977). Although a significant increase in adrenal cortical carcinomas was reported in that study, the second part of the study indicated no increased incidence of neoplasms in rats fed dieldrin at 2, 10, or 50 mg/kg (NCI 1977).

A study in Syrian golden hamsters fed dieldrin (0, 20, 60, or 80 mg/kg) in the diet for up to 120 weeks indicated no statistically significant increase in tumors (Cabral et al. 1979). Other studies in dogs (Walker et al. 1969; Treon and Cleveland 1955; Fitzhugh et al. 1964) and monkeys (Zavon and Stemmer 1975) have not provided evidence of carcinogenicity.

In an NCI bioassay, B6C3F1 mice were fed aldrin in the diet at dosages of 4 to 8 mg/kg (males) and 6 mg/kg (females) for 80 weeks. Similar groups of mice were also given dieldrin in their diet at 2.5 mg/kg or 5 mg/kg. A significant dose-dependent increase in the incidence of hepatocellular carcinoma was noted in the males of the aldrin- and dieldrin-treated groups (USEPA 1980). A working group of the International Agency for Research on Cancer (IARC 1974) concluded that dieldrin and aldrin are hepatocarcinogenic in mice. Based upon the available scientific evidence, the IARC has concluded that aldrin and dieldrin are carcinogenic in rodents, but that insufficient evidence was available for their classification as human carcinogens.

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METHYLENE CHLORIDE

Methylene chloride is widely used in paint removers, degreasing agents, and aerosol propellants; as a blowing agent in flexible urethane foams; as a process solvent in the manufacture of pharmaceuticals and food products, including the decaffeination of coffee; and as a fumigant for grains and fruits. An estimated one million workers are potentially exposed to methylene chloride or to products that contain this chemical (NIOSH 1986).

In humans, direct contact with methylene chloride produces eye, respiratory tract, and skin irritation (EPA 1985b). Mild poisonings due to inhalation exposure produce solemnness, lassitude, numbness and tingling of the limbs, anorexia, and light-headedness, followed by rapid and complete recovery. More severe poisonings generally involve correspondingly greater disturbances of the central and peripheral nervous system. Methylene chloride also has acute toxic effects on the heart, including the induction of arrhythmia. Fatalities reportedly due to methylene chloride exposure have been attributed to cardiac injury and heart failure. Methylene chloride is metabolized to carbon monoxide in vivo, and levels of carboxyhemoglobin in the blood are elevated following acute exposures. In experimental animals, methylene chloride is reported to cause kidney and liver damage, convulsions, and distal paresis. An oral median lethal dose (LD_{50}) of 2,136 mg/kg and an inhalation median lethal concentration (LC_{50}) of 80,000 mg/m^3/30 min is reported for the rat.

Methylene chloride is reported to be mutagenic in bacterial test systems. It has also produced positive results in the Fischer rat embryo cell-transformation test. However, it has been suggested that the observed cell-transforming capability may have been due to impurities in the test material. There is no conclusive evidence that methylene chloride exposure produces teratogenic effects.

Methylene chloride is currently under review by the National Toxicology Program (NTP 1984; EPA 1985a). Preliminary results indicate that it produced an increased incidence of lung and liver tumors in mice and mammary tumors in female and male rats. In a chronic inhalation study, male rats exhibited an increased incidence of sarcomas in the ventral neck region (Burek et al. 1984), however, the authors suggested that the

relevance and toxicological significance of this finding is uncertain in light of available toxicity data. Methylene chloride has been classified in EPA's Group B2 (probable human carcinogen), based upon positive results in animal studies and inadequate evidence in humans (EPA 1985b).

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